THE JOURNAL OF DENTAL RESEARCH

(Continuing the Journal of the Allied Dental Societies)

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VOLUME ELEVEN
NEW YORK
1931

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WAVERLY PRESS, INC.
THE WILLIAMS & WILKINS CO.
BALTIMORE, U. S. A.

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"DENTISTRY" AND "STOMATOLOGY"

This *Journal* uses the term "dentistry" to signify the science and art of oral health-service in all its local and systemic relationships. The meanings of words, which are not necessarily restricted to *etymological* significance, acquire expanded or new import in accordance with convenience and usage. Thus the term "medicine," which originally meant to remedy or cure, now also means *to make remedial or curative treatment unnecessary.* "Stomatology" is a synonym for "dentistry." We prefer the older term because its use respects both the history of dentistry and the efforts of those who have brought dentistry to its present status.

ENDOWMENT FUND OF THE JOURNAL OF DENTAL RESEARCH

During the past three years, the American College of Dentists, through the agency of its Commission on Journalism, has been giving special attention to dental journalism. During the progress of this study, and until decisions are announced, efforts to increase our Endowment Fund have been suspended, lest any such activity should appear to be related to the endeavor of the College to bring about, by fair and suitable means, the discontinuance of trade-house control of journals that are issued as professional magazines. The annual deficit, larger than usual because of the increased bulk of this Volume, will be liquidated as heretofore by supporters of the cause of professional journalism.

PROCEEDINGS OF AFFILIATED DENTAL SOCIETIES PUBLISHED AS INDEPENDENT JOURNALS WITHIN THE JOURNAL OF DENTAL RESEARCH

The organizations named on the title-page publish all or some of their proceedings on conditions that make each body's group of papers, in effect, its own independent journal within the *Journal of Dental Research.* Papers contributed by organizations are controlled and edited by the official representatives. Reprints of parts of any number of the *Journal* may be obtained for issuance in separate volumes of transactions. See the title page of this Volume, and compare it with those of Volumes V to X, inclusive.

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WAS THERE ANY "CONFUSION" IN THE ORAL HYGIENE COMMITTEE'S CRITICISM OF MONOGRAPH III OF THE AMERICAN CHILD HEALTH ASSOCIATION[1]

ORAL HYGIENE COMMITTEE OF GREATER NEW YORK[2]

A preprint of the preliminary comment by Dr. Palmer,[3] on our criticism of the American Child Health Association's Monograph Number III,[4] was presented by the *Journal of Dental Research* to our Committee at its monthly meeting on November 18, 1930. The only remarks in Dr. Palmer's preliminary comment that appear to require response are his reference to "obvious confusion" in our protest, as indicated by a purported failure therein "to distinguish between the publication of research findings and the announcement of an Association policy;" and also his statements that "Monograph III is published as a strictly technical presentation of the findings of a research and not as a recommendation of public health policy," and that "Monograph III . . . . expressed interpretations only of the dental materials as gathered"—all on page 656. These quotations might suitably be contrasted with various statements in Monograph III, but it will be sufficient to compare them with the following, as recorded on pages 1–3 of the Monograph, where purposes were explained at the outset:

"The analyses contained in this volume . . . . are undertaken in order to throw light upon problems of *health administration in the schools of the country*. . . . . *Recommendations for school health policy* . . . . sometimes must diverge considerably from current practice. . . . . One section of this Monograph deals with the effect upon decay in permanent molars

[1] The succeeding paper, presenting Dr. Palmer's complete reply to the Oral Hygiene Committee's criticism in our issue for October 1930 (x, p. 497), was received on December 18, 1930, the day on which this issue was closed, too late for presentation at the monthly meeting of the Oral Hygiene Committee on December 16, 1930.

[2] Sub-committee on Research Monograph III: H. Shirley Dwyer, Thaddeus P. Hyatt, William J. Gies, Kannon Sheinman, and Maurice William.

[3] Palmer: *Journal of Dental Research*, 1930, x, p. 655.

[4] Oral Hygiene Committee of Greater New York: *Ibid.*, 1930, x, p. 497.

exercised by filling of deciduous molars. *We do not present this treatment of our materials with the hope of adding any exact knowledge in this field.* . . . . We are, however, *faced* with *propaganda directed at public health administration* and claiming that a *policy* of filling deciduous teeth will protect the first molars against decay. (*Author's footnote:* These claims are made with regard to the *frequent occlusal caries* and not only to the infrequent decay on the sides of the permanent molars.) *Our data are organized to critically examine such advice and are here presented in order that public health administrators may be equipped to judge the value of these arguments.* Data of this kind . . . . may . . . . be very important if they indicate that in undertaking the widespread filling of deciduous teeth *the motives of protection against subsequent caries in the permanent molars must be excluded. Administrative practice* must be able to ascribe to each procedure its appropriate results in order to *build* a well integrated *school health program.* . . . . It is not only necessary to know the truth, it is necessary to have convincing evidence which will lead to *correct practice on the part of administrators and will protect them against specious argument from uninformed or prejudiced sources.*" (Italic not in the original.)

In the fourth sentence of the foregoing quotation from Monograph III, its author states very definitely that Monograph III is *not* "a strictly technical presentation of the findings of a research." In various portions of the same quotation we find "a recommendation of public health policy." In each of these instances, which are here presented only as illustrations, the facts indicate just the opposite of the claim, by Dr. Palmer, that in our criticism of Monograph III we failed, owing to "obvious confusion," to draw what he regards as an important distinction.

The Oral Hygiene Committee, referring to statements in the above quotation from Monograph III, protested against "the pretended existence of responsible 'propaganda' for filling deciduous teeth to protect the *occlusal surfaces* of first permanent molars against decay," and added that this "unwarranted and misleading statement," which was *not* an "interpretation only of dental materials as gathered," is "unjust to the dental profession, and wholly unworthy of any serious scientific research" (page 503). In the Oral Hygiene Committee's protest it was also indicated (page 545) that the Mouth Hygiene and Preventive Dentistry Section of the American Dental Association stated, in a series of resolutions, that "no dental propaganda carried

on by any of the official dental educational organizations has ever advocated such a policy." The author of Monograph III said (page 2): "we" (presumably the public or the American Child Health Association) are "faced with propaganda." "*Faced*" indicates an exigent situation, and "propaganda" means something more than casual comment or unsupported remarks. The assertions in Monograph III relating to this exigent situation, and to "propaganda" in behalf of a policy of filling deciduous teeth to protect the *occlusal surfaces* of first molars against decay, have raised serious doubts among dentists regarding the sincerity and reliability of the Monograph. These doubts were clearly stated in our "Quotation 1" on page 503,[4] where our most comprehensive objection was indicated unmistakably. This particular criticism, we feel, should not have been ignored, but rather should have had first attention, in any published reply by a representative of the American Child Health Association intended to promote understanding, for the untruthful statements in Monograph III about this imaginary "propaganda" will continue, by implication at least, to disparage the intelligence, knowledge, and disinterestedness of the whole dental profession wherever Monograph III is accepted as a responsible publication, and until these misleading statements are publicly disavowed by or on behalf of their author. We regret that the Director of the Division of Research of the American Child Health Association did not include in his "preliminary comment"[3] either a substantiation of the alleged existence of this "propaganda," or a candid withdrawal of the invention in Monograph III relating thereto. This, we believe, could have been done very easily during the interval between September 23, 1930, and October 18, 1930, which Dr. Palmer mentioned in the first two sentences of his "preliminary comment" as the time available for the preparation of that comment.

Addendum[5]

A copy of the succeeding complete reply,[6] by Dr. Palmer and his associates, to our original protest was received in time to enable us to

[5] Copies of the foregoing response by the Oral Hygiene Committee were submitted by the *Journal of Dental Research* to Dr. Palmer for the authors of the succeeding paper, and vice versa, so that brief addenda could be appended to the printer's proof, if desired, to simplify the further discussion. Dr. Palmer added an introductory note (page 7); the Oral Hygiene Committee appended this Addendum.—[*Ed.*]

[6] Palmer *et al.*: *Journal of Dental Research*, 1931, xi, p. 7.

append this Addendum, which, made as brief as possible, is restricted to the subjects of the foregoing comment.

We have emphasized the fact that Dr. Palmer, in his "preliminary comment," ignored the first and most important matter in our protest; namely, "the pretended existence of responsible 'propaganda' for filling deciduous teeth to protect the *occlusal surfaces* of first permanent molars against decay" (p. 503). In the succeeding complete reply we find the following admissions regarding the invented propaganda (page 14):

". . . . our Monograph does not say [*sic*] that this 'propaganda' is put forth by dental organizations. This is an interpretation introduced by the Oral Hygiene Committee. We admit that the Oral Hygiene Committee has reason for criticising our use of the word 'propaganda.' Instead of 'propaganda' we might [*sic*] have said 'unwarranted claims.'"

We accept this statement as evidence that none of the comment about "propaganda" in Monograph III was warranted, and that none of it should have appeared in what Dr. Palmer has called a "strictly technical presentation of the findings of a research." The failure of Dr. Palmer, in his "preliminary comment," to accept the earliest possible opportunity to absolve the dental profession from all responsibility for the "propaganda" that was invented in Monograph III, has been made impressive in its significance by the manner in which this belated admission has been recorded and qualified. The admission that the protested "propaganda" never existed is preceded and followed by statements so worded as to beguile the unwary reader into believing that, in this relation, the Monograph was not so far out of the way after all. Not only is the nature of the alleged "propaganda" misstated, but the citations (A-E) intended to justify the Monograph are irrelevant. Two sentences from Dr. Palmer's statement will suffice to show the evasions:

"Our Monograph made reference to the existence of propaganda directed toward public health administration which claimed that the filling of deciduous teeth *prevented decay in the permanent teeth.* . . . . Actually the idea that the filling of deciduous teeth *does protect the permanent teeth against decay* has been endorsed by responsible dental organizations in so far as the expression of this thought in health education materials, or in their annual

reports, may be considered an endorsement" (italic not in the original). (Pages 13 and 14.)

In these assertions Dr. Palmer suppresses the claim in the Monograph that the alleged "propaganda" included "*frequent occlusal caries*" (Monograph, p. 2). The citations (A-E), on pages 14–16 that follow the misstatements quoted above, miss the point at issue; namely, "propaganda" claiming, as asserted in the Monograph, that the filling of deciduous teeth specifically protects the *occlusal surfaces* of permanent molars against decay. That the filling of deciduous teeth may prevent "*infrequent* decay on the *sides*," as implied in the Monograph, is not in any sense involved in our discussion. The distinction in the Monograph between "infrequent decay on the *sides*" and "frequent *occlusal* caries," and the import of this distinction, are self-evident. Therefore we cannot assume that the distinction was ignored, and the point at issue misstated, with any intent to enlighten the reader or to promote understanding.

The analysis of our original protest, by Dr. Palmer and his associates, contains many statements indicating inattention to the phraseology of our comment. Accordingly, we call special attention to this remark in their reply (page 9): "In preparing our answer to the protest of the Oral Hygiene Committee we shall present the main elements of this controversy as a self-contained document *making it unnecessary to refer back to the original documents*" (italic not in the original). If the reader did not "refer back to the original documents," the evasion indicated above would not be discovered by him. We suggest that readers of the reply should carefully "refer back to the original documents" in every instance of disagreement.

In a succeeding issue we shall refer to matters in the reply that range beyond the scope of the foregoing remarks.

PUBLIC HEALTH ASPECTS OF DENTAL DECAY IN CHILDREN: RESEARCH MONOGRAPH III[1]

A Reply to a Protest Against This Monograph by the Oral Hygiene Committee of Greater New York[2]

George T. Palmer, Dr.P.H., *Director*, and Raymond Franzen, Ph.D., Anne Whitney, Harold H. Mitchell, M.D., C. Mahew Derryberry

School Health Study Staff, American Child Health Association, New York City

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I. NOTE

In submitting this reply to the "protest" of the Oral Hygiene Committee of Greater New York re Monograph III, the American Child Health Association realizes that misunderstandings have arisen due to misinterpretation of the technical terminology employed and to shortcomings in appreciating the viewpoint of the dental profession. In the "reply" a serious effort has been made to clarify the language and intent of Monograph III, to correct its shortcomings from the standpoint of the dental approach, and to explain the American Child Health Association's contentions with reference to controversial points on which it believes its position is sound. The undersigned believe the American Child Health Association is sincere in its endeavors to build up a constructive program for the conservation of child health. We ask respectful consideration of this "reply," and suggest that any differ-

[1] Research Monograph III, "Public health aspects of dental decay in children," was published by the American Child Health Association in March 1930.

[2] Oral Hygiene Committee of Greater New York: *Journal of Dental Research*, 1930, x, p. 497; October.

ences that may then remain unsettled be harmonized at a round-table conference rather than by further efforts through published statements.

(*Signed*): FINN J. BRONNER, MILO HELLMAN, JOHN OPPIE MCCALL, JACOB SCHNEER, ALFRED WALKER

II. INTRODUCTORY

Preceding publications. In March 1930 the American Child Health Association published an account of some original research under the title: "Public health aspects of dental decay in children."[1] In the October, 1930, issue of the *Journal of Dental Research* the Oral Hygiene Committee of Greater New York published comments on the above research under the title: "School Health Research Monograph Number III of the American Child Health Association, entitled, 'Public health aspects of dental decay in children:' an analysis, a protest and a correction."[2] To this lengthy protest the American Child Health Association made a very brief reply in the December issue of the *Journal of Dental Research*,[3] and on that occasion promised a more extended answer later. The present statement is this more extended answer.

The gist of the argument. Monograph III is an original research whose purpose is to develop helpful information for the administrators of public dental clinics such as school clinics. In this Monograph we have presented evidence to show that the enamel defects which are most dangerous are the ones that are most difficult to detect. Consequently a public health or clinic policy of filling pits and fissures as it is now advocated is not the best kind of prophylactic odontotomy. We contend that a method of distinguishing pre-carious enamel defects is much needed. We believe that there is a great deal of support of this thesis amongst the dental profession. The Oral Hygiene Committee does not meet this contention, but maintains that we are defending another proposition entirely. It argues as if we had stated that enamel defects have no effect on caries. We have no reason for taking such a position. Our Monograph contains evidence regarding *the detection of liability to caries.* It makes no pretence to present morphological evidence. It is a contribution to diagnostic method, not to etiology.

The intent of Monograph III. It is most important to keep in mind,

[3] Palmer: *Journal of Dental Research*, 1930, x, p. 655; December.

as stated above, that Monograph III is a research publication and that its findings have their application to public dental clinics. Monograph III is not a review of the field of other research, nor a recommendation of the administrative policy of the American Child Health Association. It is specifically stated that the applications of this research do not relate to the private dental practitioner. These points have been emphasized in the Monograph, in the Foreword and in Chapter I.

In preparing our answer to the protest of the Oral Hygiene Committee we shall present the main elements of this controversy as a self-contained document making it unnecessary to refer back to the original documents.

III. BACKGROUND OF MONOGRAPH III

Before proceeding to the consideration of the items of Monograph III criticized in the protest and our reply to these criticisms, we wish to establish a broad foundation for this discussion of the question of dental service for children. We have studied the dental literature both in its scientific contributions and in its popular presentation to school teachers and the laity. We have made surveys of the publicly and privately supported health agencies in communities, many of which included dental service in public schools. These inquiries have made us aware of the differences of opinion existing among the dental profession on such subjects as the mechanism by which caries starts; the means by which caries may be prevented; and the general type of school dental program that is most effective.

When the opportunity presented itself to us of studying intensively certain limited phases of the effectiveness of health programs in the public schools, we decided that mouth conditions were an important feature to be included in that study. This decision was strengthened on being advised by dentists that many statements accepted as facts by a large proportion of the profession were not based on scientific evidence. Information based on such research could be placed at the service of official and private agencies charged with public health administration of a public dental program.

We believe that oral disease, especially root-end infection, caries, and the premature loss of both deciduous and permanent teeth are

serious handicaps to the health of children. Therefore we are enough concerned with the cause of the dental care of children to have a vested interest in the widespread adoption of a program once it is proven to be effective and practical. Public health officials are just beginning to assume an appropriate obligation in the problem of oral disease in children, and it is certainly to the best interest of the dental profession that the dental part of school health programs shall be based on the best available information.

Briefly then, we believed that the teeth of school children should be cared for as a health measure; that there was a real need for the various studies presented in Monograph III; and that these studies, even though necessarily limited in their scope, would play an important rôle in setting up a public dental program. We may add that, as announced in our preliminary comment,[4] we "hope in the *future to express an Association attitude on dental service in the schools in its administrative aspects.*" (Italic not in the original.)

IV. PROTEST OF THE ORAL HYGIENE COMMITTEE

The substance of the Oral Hygiene Committee's published protest is that our technique of measuring mouth conditions was faulty and our interpretations of the data thus gathered were unfounded. Further than this there is the charge that our intent was destructive and not sincere. As to this latter charge, we submit that the very foundation on which our Association rests and its works over the years are contradictory to any such an assumption. Monograph III is openly clothed in the language of research, a language that is precise, that addresses itself to the points at issue. Its purport, its nature and its contents are clearly and unmistakably labelled as research. We contend that research, distinctly so marked, cannot be obstructive, even though its conclusions should conflict with previous research, clinical experience or prevailing professional opinion.

Does it help the scientific discussion of this question when the Oral Hygiene Committee uses such phrases as these in its published protest? (italic not in the original):

[4] Palmer: *Journal of Dental Research*, 1930, x, p. 656; December.

"The Monograph is almost wholly destructive in *intent* and presentation."

"There is a suspicious selection of data to support *favored* conclusions—all savoring of *prejudice*."

"Essentials of procedure have been *omitted* from the record."

"The *pretended* existence of responsible propaganda."

"*Presumed* mathematical accuracy."

"A *supposedly unprejudiced* study."

"Was this gross departure from the scientific method due to *accident*, or to *carelessness*; or did the excluded data prove something contrary to *predetermined* conclusions?"

"The facts in this paragraph of our comment do not support a belief that the Monograph is an accurate and *sincere* presentation."

"If the Monograph had been intended to avoid constructive suggestions, to misrepresent current dental practice, and to degrade dentistry in public confidence, it would have been difficult to devise it more *adroitly* for the achievement of these aims."

We are disappointed that the Oral Hygiene Committee has in its protest indicated no clear insight into the purport of Monograph III, nor has the Committee presented any evidence which might lead to qualifications of the conclusions in the Monograph, or to doubt of the interpretation of the findings expressed therein.

V. ANSWER TO THE PROTEST

Following the table of contents in the Oral Hygiene Committee's protest, and the two pages of an abstract of the report and two pages of introductory matter, we come to page 502, on which detailed criticisms begin. Three points are made. The *first* criticism (page 502) is that the title of our Monograph, "Public health aspects of dental decay in children," is inappropriate. The preliminary title chosen was "Factors in dental decay." In our discussions with dentists over the preliminary mimeographed copy it was brought out that our research related to dentistry done for groups of children in the schools or in clinics outside the schools, where demands for service were too heavy to permit the dentist to have an intimate knowledge of the previous history and present characteristics of his patients. Our research thus did not necessarily have a bearing on the work of the private dental practitioner who, from his detailed knowledge of each individual case, may

be expected to follow a course of action appropriate to that individual. One of our reviewers, in an English publication,[5] appreciates this distinction when he writes:

"This monograph, which is one of a series published by the above association, is an attempt to evaluate some of the factors connected with dental hygiene which may have a bearing on the *public health* aspects of dentistry. In this sense it is significant, for though much has been written on the dental treatment of school-children there is so far little realization that *public dentistry is not private dentistry on a larger scale*, but involves different methods of approach, just as the tuberculosis problem as it affects the individual differs from the same problem affecting the population as a whole." (Italic not in the original.)

We therefore modified the title to read: "Public health aspects of dental decay in children." The words "public health" were intended to imply the public clinic considerations of the study in distinction from the interest of the private dental practitioner. The word "aspects" was included to indicate the limitations of the Monograph, implying that only certain parts of the large and important problem were considered in this study, and not in any way to imply that all aspects were considered.

The *second* criticism (page 502) is that we confined our remarks to our own study and did not refer to other researches along this line. This Monograph was Number III of the School Health Study Series of Monographs. In Monograph III we presented original findings derived in the course of a larger study whose object was to find a means of measuring the effects of school health programs on children. We were not writing a review on the subject of preventive dentistry nor on the various studies that contributed to the policies followed by various school dental programs.

In the same section of the protest (page 502) we are taken to task in the following words referring to the author of our Monograph: "He occasionally admits that his views and data are of importance to school health administration, the present policies of which he *intimates should not include dental service.*" Nowhere in our Monograph is there the slightest intimation that dental service should not be

[5] *Lancet*: 1930, ccxix, p. 747; October 4.

included in a school health program. The Oral Hygiene Committee is wholly unjustified in making this statement.

The *third* criticism (page 502) is that our results conflict with reality, scientific knowledge and clinical experience. We are aware that our data and interpretations conflict with the views of individual dentists. We question, however, whether such a disagreement provides grounds for assuming that our data and interpretations are in conflict with reality and scientific knowledge. We have presented our evidence and our methods. Although in the presentation of our own findings we have made no reference to other research or clinical experience, we have not ignored such evidence in making our interpretations and drawing conclusions. Much dental literature and many dentists have made available a wealth of information bearing upon the problem. If research led to no new ideas, it could hardly result in progress. Scientific research invites open-minded criticism, giving due consideration to data, analysis, and interpretation.

Pages 503 to 533 of the criticism are devoted to a discussion of 15 quotations from the Monograph. We shall take these up in order.

Comment on quotations 1–15 of the protest

Quotation 1, page 503. Our Monograph made reference to the existence of propaganda directed toward public health administration which claimed that the filling of deciduous teeth prevented decay in the permanent teeth. The Oral Hygiene Committee pokes fun at our assertion and says in effect that we have set up a straw man to knock him down in order to gain the approbation of our readers. The criticism further states (a) that our action tends "to destroy the confidence of public-health administrators in public-health-education propaganda as actually conducted by dental organizations;" (b) that this "is unjust to the dental profession;" (c) that this "ludicrous view. . . . has never been endorsed by responsible dental organizations." The Oral Hygiene Committee, in its protest, gives three reasons for a policy of filling deciduous teeth with which we have no disagreement. The point we make in the Monograph is that the fillings in deciduous teeth exercise no important influence upon the decay of the occlusal surfaces of the permanent teeth. This point of

view is very clearly appreciated in an English editorial[6] regarding this controversy, which reads as follows:

"In the monograph we have as a proposition which is proved: 'Filling deciduous teeth has no apparent effect upon the subsequent or contemporary decay of permanent teeth.' The conclusion from this is that filling temporary teeth to prevent decay of permanent teeth is useless. The dentists object to this upon three counts, not one of which is relevant to the question at issue; for they are all reasons, other than the one under consideration, for filling deciduous teeth. Whether it is wise to fill temporary teeth is a very big question; whether it is wise to fill them to prevent decay of the teeth which will displace them is one small part of that question, and to this part, and only to this part, the monograph gives a negative reply. The dentists may retort that they never suggested filling temporary teeth for this specific purpose, but it has been given as a reason for preventive dentistry and it is of value to know its exact relevancy."

In the next place our Monograph does not say that this "propaganda" is put forth by dental organizations. This is an interpretation introduced by the Oral Hygiene Committee. We admit that the Oral Hygiene Committee has reason for criticizing our use of the word "propaganda." Instead of "propaganda" we might have said "unwarranted claims."

But the Oral Hygiene Committee states that this "ludicrous view. . . . has never been endorsed by responsible dental organizations." Actually the idea that the filling of deciduous teeth does protect the permanent teeth against decay has been endorsed by responsible dental organizations in so far as the expression of this thought in health education materials, or in their annual reports, may be considered an endorsement. Evidence on this point is as follows:

A. In mimeographed material entitled: "Better teeth: better health,"[7] stories compiled and distributed by the American Dental Association, Department of Dental Health Education, appears the following: "Take good care of your first teeth or you may have poor second teeth."

B. There is a pamphlet in print, "Better teeth: better health"[7]—

[6] *Medical Officer*, 1930, xliv, p. 201, November 8.

[7] This material, we understand, was prepared by the Department of Dental Health Education prior to the present administration of the Bureau.

songs and rhymes published by the American Dental Association – which contains this rhyme (italic not in the original):

"Little teeth are moving over
Larger ones are going to come
They are rooted deep and waiting
Hidden just beneath the gum.

"Wise wee boys and girls are working
With their tooth brushes every day
So the mouth and gums are ready
For these new teeth come to stay.

"If they find all sweet and healthy
But if not, why right away
They grow sore and lose their beauty
Start to *crumble and decay.*

"So be very, very careful
Guard your mouths with greatest care
For the other teeth are coming
They will soon be there."

C. In the January, 1916, report of the Forsyth Dental Infirmary, page 4, "Objects of the Institution," the second object was stated thus: "To prevent dental caries, by oral prophylaxis and by the care and preservation of the temporary teeth." This statement also appeared in the 1918 report. In the Sixth Annual Report the following statement is made: "The temporary teeth, not diseased, are also given much attention on account of their influence on the regularity and *soundness* of the permanent ones." (Italic not in the original.) This same statement appears in the subsequent reports for 1922, 1923, 1924, 1925, 1926, 1927, and 1928. In the reports for 1929 and 1930 we do not find this statement. Instead we find this expression, which is quite different: "The importance of retaining the temporary teeth for the proper growth and development of the maxillary bones is recognized and destruction of these teeth is limited as much as possible." Further evidence that this is not a "ludicrous view" follows in D and E:

D. In commenting on the published conclusions in our Monograph, on page 39—which reads: "Filling deciduous teeth has no apparent effect upon the subsequent or contemporary decay of permanent teeth"—one of the prominent members of the Mouth Hygiene and Preventive Dentistry Section of the American Dental Association wrote us that we are wrong in this conclusion indicating that he thinks that the filling of deciduous teeth does protect the permanent teeth against decay.

E. In the *Dental Cosmos* for April, 1927—a study by Marion R. Stevens, B.A., D.D.S., entitled: "Significance of caries in deciduous teeth in relation to structural formation of enamel of first permanent molars at time of eruption"—one of the author's conclusions favors prompt attention to the first permanent molars because of the relation of caries in the deciduous teeth to a parallel condition in the first permanent molars. This study was made under a grant of the Research Commission of the American Dental Association.

Quotation 2, pages 504 and 505. In this instance we are criticized for drawing the conclusion from our data that "present dental correction is palliative and not either preventive or curative," and that "a real preventive program awaits solution of the problems of predisposition and cause." The Oral Hygiene Committee maintains that this statement will, if allowed to stand uncorrected, seriously retard the progress of oral health service for children, mislead school administrators and misrepresent the capabilities of modern dentistry. This conclusion, in context with the three statements summarizing the data on which it is based,[8] does not have a bearing on the capabilities of modern dentistry. It has reference to prevention in the strict sense, that is, the prevention of caries as a disease problem. It does not deny the value of "extension for prevention" as a practice of operative dentistry, nor does it refer to the effect of recurrent decay. Obviously it supports the practice of early filling of caries to prevent its deeper penetration. By palliative was meant the lessening, abating or mitigating effect of fillings upon caries. Palliative was used to mean conservation. The distinction between conservation and prevention have been very well illustrated in an editorial of the *Bulletin of the First District Dental Society of the State of New York* for May 1930.

Nor are we alone in this view as expressed. For example in the

[8] See the Monograph: Chapter 3, and the summary on page 39.

September, 1930, issue of the *American Journal of Diseases of Children* appears an article entitled, "Problems of dental caries," by Bunting, Hadley, Jay, and Hard where they have this to say (page 536):

"For up to the present time no practical means of preventing dental caries has been offered, other than that of oral cleanliness which is of doubtful efficiency. The discovery of some definite means of controlling or preventing this highly prevalent and important disease would be an inestimable boon to mankind."

Bödecker also writes:[9]

"Science must and will find the definite cause and the manner of prevention of dental caries. Until, however, this Utopia is reached, all that most of the dental profession can do at present is to repair the ravages of decay."

Study of Chapter III of our Monograph reveals that we have presented definite evidence that current dental service does hold back dental caries and conserves the teeth against the destruction wrought by this disease. This is clearly stated in one of the summarizing statements of the chapter where we say: "The chances for further development of caries in any one tooth are smaller when previous decay in that tooth has been corrected." This chapter, therefore, supports the practice upon which we all agree that early care and attention by a dentist conserves children's teeth against the ravages of caries. The above conclusion refers to total amount of caries, which is a product of number and degree. (See the Monograph, pages 77 and 78.)

However, we find that the Oral Hygiene Committee wishes us to consider prophylactic odontotomy (page 505) and its possibilities. Chapter III under discussion makes no attempt to contribute to an evaluation of prophylactic odontotomy unless the Committee wishes to claim that *current dental practice* is now characterized by such a widespread application of prophylactic odontotomy as to be represented by the conditions found in the children's teeth examined by our dental hygienists. Our judgment would be that the part of current dental practice which is considered in Chapter III is much more a practice of cutting out and filling more or less advanced carious cavities than

[9] Bödecker: *Dental Cosmos*, 1929, lxxi, p. 286; March.

it is a practice of filling non-carious enamel defects. Too often children get no dental care until caries is well advanced and it is this problem that motivates the subject matter of this chapter.

In this connection the Oral Hygiene Committee offers an analogy between caries and cancer. We quote several sentences bearing on this point (page 505): "There is useful medical propaganda (a) to show, from data obtained in a study of adults, that various lumps, swellings, tumors, etc. although seemingly unimportant departures from normal, tend to evolve into cancer, and (b) urging that physicians be consulted regarding them so that these 'growths' may be surgically removed *before* this pathological change can begin. In such cases, surgeons, in order to remove *all* 'growths' that would *probably* become cancerous if left in place, are obliged also to excise *some* that *might* remain completely harmless if undisturbed, thus engaging in 'operative interference on many units merely because it prevents disease in some,' which the author concludes 'is not sound policy' (page 49)." But "pre-cancerous" growths are certainly abnormal. They are not so common as are pits and fissures. Bödecker writes:[10]

> "Fissures are so frequent in the occlusal surfaces of molars that they may well be regarded as a common occurrence; yes, even a *normal condition*."

Of course, under conditions of private practice moles and other minor growths may be surgically removed—"some that might remain completely harmless if undisturbed." But we know of no public health program against cancer which provides for the systematic examination and the surgical removal of all moles and minor "growths," on large population units such as we have reference to in our Monograph in considering the application of prophylactic odontotomy to public clinics. Even in periodic health examination clinics the advice in regard to "lumps, swellings and tumors" is to refer the case to a surgeon who will consider the history of the development of the suspected neoplasm under better conditions for careful discrimination than is practicable in a routine examination in a public clinic.

The Oral Hygiene Committee under this discussion seems to disapprove of our opinion expressed in Monograph III, Chapter IV,

[10] Bödecker: "The toothbrush in relation to occlusal fissures," *Dental Items of Interest*, 1926, xlviii, p. 163; March.

when we refer to a policy that would "cut out and fill all the developmental grooves in the occlusal surfaces of first molars" as an unsound policy.[11] Does the Oral Hygiene Committee favor a public clinic policy of cutting out and filling all the developmental grooves in the occlusal surfaces of first molars? Or does the Oral Hygiene Committee favor only the cutting and filling of the recognizable non-carious enamel defects as soon as the tooth is erupted? If only the latter policy is favored, should this policy be applied only to private practice conditions and such other conditions as are suitable for experiment and research, or should the policy be extended to public clinics so as to be sure to reach great numbers of children who have non-carious enamel defects? If this latter public clinic policy is what the Oral Hygiene Committee approves, then we shall continue to insist that we have research data bearing on the problem until other scientific facts show we are wrong. If the Oral Hygiene Committee is not concerned with the defense of prophylactic odontotomy in public clinics and its extension on a widespread scale, then we should be able to overcome our supposed differences.

Quotation 3, page 506. On pages 1 and 2 of our Monograph appear the following statements: "There is no attempt to confirm or deny decisions which are primarily the prerogative of the dental profession nor to verify or dispute the findings of those scientific branches of knowledge upon which decisions of this profession rest. Recommendations for school health policy, however, sometimes must diverge considerably from current practice." The last sentence in this quotation forms the basis of criticism No. 3. The protest states that where health is at stake the obligations of public clinics and private practitioners are identical.

The distinction between what can be carried out practically in a public clinic and what may be reasonable under conditions of the private practitioner is evidently not appreciated by the Oral Hygiene Committee. In private practice, where the dentist has an opportunity to know something about the individual child, he may consult with the parent and carry out special measures that are impractical in a public program of health service for all children. It is expected

[11] Pages 48 and 49 of Monograph III.

that he may know something about the susceptibility to caries of his individual patient. He can consider the response of the patient to treatment measures that he may recommend. He may apply extraordinary measures for examination of the teeth by x-ray, repeated observations and instrumentations. In short he may exercise discrimination and observation that would be quite out of the question under a public clinic policy of filling all enamel defects. It is well recognized among public health workers that such individual and discriminating policies as continued observation and inquiry into systemic and constitutional factors have not yet been possible in a program of general public health service. The large number served requires rapid and routine methods and little variation of procedure to meet individual needs.

We believe that the Association's School Health Study Staff might appropriately present their evidence with reference to a policy in public clinics without the insinuation in the protest that we stand for the following (page 506): "A policy passable today would also be acceptable years hence—nothing should be done to effect improvement; things as they are might be worse; adopt the easiest way, not the most desirable method." We are criticized for not trying to show in our Monograph what should and could be done in a public dental health program. The Monograph made no attempt to deal with this question, not because it is unimportant but because this Monograph was not devoted to that subject.

Quotation 4, pages 506–9. Here are three pages of comment in which the Oral Hygiene Committee gives their version of what was meant in our Monograph, likens the fillings of pits and fissures to smallpox vaccination, states the gist of our thesis in a most satisfying manner, and then chides us for not endorsing the universal filling of all pits and fissures.

First the Committee gives a quotation from our Monograph as follows: "It may be proper policy for a dentist to examine, cut out, and fill the grooves [sic] on the occlusal surfaces of the first molars no matter what criteria he uses to distinguish teeth that shall be so fortified. It is, however, definitely bad public health policy to inaugurate a widespread practice of this kind unless it is possible to lay down a definite manner of finding those occlusal surfaces of first molars

which are liable to caries and those others which are not (page 2)." In the second line of the above quotation, we omitted the word "developmental" before grooves. The Committee point out our mistake in expression but they go too far. They continue to say that by grooves in the above quotation we probably refer to "pits and fissures." We do not refer to pits and fissures; we refer to developmental grooves. The Committee then states that we use "occlusal surfaces" in one place to mean defects or faults in occlusal surfaces. The Committee is mistaken. By occlusal surfaces we *mean* occlusal surfaces.

It may be seen from this quotation (No. 4), and its context in the Monograph, that it is given as *an illustration* of a possible difference between private practice and a public clinic policy. This difference we have already discussed. This illustration might have been explained as follows: The private dentist *might*, for example, in children extremely susceptible to caries, put fillings in the occlusal developmental grooves. On the other hand to inaugurate such a widespread practice as cutting out and filling developmental grooves would be bad public clinic policy unless the operator had some criterion for judging which occlusal surfaces of the teeth were likely soon to harbor decay. This idea was suggested to us by Dr. Thaddeus P. Hyatt in one of our numerous conferences with him. Dr. Hyatt, as we understood it, had considered the policy of fillings for all occlusal developmental grooves regardless of the susceptibility of the child and had rejected it in favor of filling only the enamel defects that can be found with a fine explorer.

The Oral Hygiene Committee next presents the following (page 508): "Regarding the practicability of recognizing dangerous enamel defects, and its relation to a school program of prevention, we call attention to well known facts. Some enamel defects may be so minute that they cannot be easily detected, but their proportion is so small, compared with the larger number that can be, that it would be inexcusable to do nothing about those that can be found." In this statement the Committee comes very near to facing the problem presented in Chapter 4 of the Monograph, but they dodge the issue by relying on what they call "well-known facts," the issue being whether it is practical under public clinic conditions to recognize the dangerous enamel defects. Of course, we should agree it would be

desirable. The Committee mentions the enamel defects so minute that they cannot be easily detected, but it presents no evidence that these enamel defects are rare or infrequent. Must we rely upon this assertion? It is no reflection upon the Committee's clinical experience to question whether this assertion is scientific evidence. Medicine has been misled too many times by clinical experience for us to rely upon it as final proof.

The Oral Hygiene Committee appears to dispose of the problem which interests us by asserting their conviction as follows (page 509): "As for the difficulty in making the distinctions mentioned in Quotation 4 [recognizing dangerous enamel defects], and its assumed insurmountability, it will be sufficient for this report to add that procedures for this diagnosis have been conducted successfully in public schools not only by the Oral Hygiene Committee of Greater New York, but also by many dental authorities." With all due respect for the ability of members of the Oral Hygiene Committee, we ask for evidence on this point. This is the crux of the whole matter, which the Oral Hygiene Committee brushes aside with an assertion but without any evidence. The difficulty of recognizing the enamel defects with minute openings has been emphasized by Bödecker:[10]

"Figure 4 shows a type of groove in a molar that is exceedingly treacherous on account of the inability of the dentist to recognize the existing conditions. The operator might probe such a tooth with the utmost care, and not be able to detect any place where the instrument would catch. Our finest explorer is about equal in diameter to the fine toothbrush bristle (18 μ) shown in the photomicrograph. We can therefore readily understand that the dentist could not discover the minute crevice leading into the depths of the enamel. A section through the tooth, however, proves that, even though externally the groove appears to be almost self cleansing, a minute crevice extends halfway to the dento-enamel junction. This is seen as a delicate white line marked (X) and may be considered as a typical fissure, i.e., the absolute failure of the lobes of the enamel to coalesce. The section is evidently taken from a tooth of a young individual because the signs of abrasion are slight. The carious process, therefore, has not had time to reach the dentin."

Under "*Conclusions*" in this article, Dr. Bödecker makes the following statement:

"Therefore, as over 98 percent of the first permanent molars do become carious, be it resolved to place small fillings in the grooves of these teeth, irrespective of whether they are carious or non-carious, as soon as practicable after eruption."

On page 508 of their protest, the Committee states another "well known fact": "Not every enamel defect becomes carious, but the majority that will certainly soon harbor decay is so large that, to prevent caries in these, an operative preventive procedure relating to them is necessary where health is the primary consideration." This is certainly a controversial point and should not be used as a sweeping refutation of interpretations based on evidence. An entirely different point of view is contained in an article by C. N. Johnson, D.D.S., entitled, "Preventive dentistry: its scope and possibilities," *Journal of the American Dental Association*, February, 1930. He writes:

"In very susceptible mouths, no precaution is too great to protect the teeth against the progress of decay, and, in these mouths, a fissure in a tooth must always be looked on as a menace and should be cared for. Filling, of course, under these circumstances, is the most certain method of protecting the tooth, and in extreme cases, there is no question of its desirability.

"But in very many cases, I do not consider this practice justifiable if decay has not yet begun in the fissure. In children who are immune, or who promise an early immunity, I have never yet been able to bring myself to put a drill in an otherwise sound tooth and open a fissure. I have seen too many cases in which such teeth have gone without decay for a lifetime to justify me in anticipating trouble by drilling every fissure and inserting a filling. As I have intimated, I would base my decision largely on the evidence of susceptibility in that particular mouth."

We gather from the protest that the Oral Hygiene Committee of Greater New York recommends a widespread public clinic policy of filling all pits and fissures. Two large difficulties develop in regard to this recommendation. One is the problem of immunity. The other is the strong probability that the danger lies in the minute enamel defects which are very difficult to detect. Both of these difficulties are discussed in our Monograph. And it is these discussions which have aroused the Oral Hygiene Committee to their protest. We have excellent support for both. Dr. Johnson in his recent article in the

Journal of the American Dental Association definitely avows the importance of consideration of susceptibility before operative interference. Dr. Bödecker repeatedly asserts that there exist some dangerous enamel defects which are very difficult to detect. The public health implications were not discussed since their articles refer mainly to private practice. But their publications do show that the basic conclusions from our data are consistent with well informed opinion in the dental profession.

An analogy used by the Oral Hygiene Committee in this connection runs as follows (page 508): "The dental profession believes that adequate diet and hygienic living greatly reduce the incidence of dental caries *and of some other diseases as well. Perhaps* adequate diet and hygienic living would make vaccination unnecessary. But this remote 'Perhaps' would not justify discontinuance of vaccination as a general means of prevention—a means which, because of present uncertainties, must be applied to all in a community to protect each of those who otherwise would *probably* have smallpox even though some *might* not." There are two faults with this analogy. First, other factors which are involved in the development of caries are certainly far more pertinent than diet is to smallpox. Second, vaccination is a demonstrated success as a community hygiene measure mainly because of the feature of contagion, and this is not involved at all in the question of which teeth shall be operated upon to prevent caries. Vaccination of many to prevent occurrence in a few is sound policy in the case of smallpox just because a single occurrence is a liability to the whole community. It does not follow that all enamel defects should be filled because some of them grow carious. If smallpox were not contagious the importance of distinguishing the immune from the susceptible would be apparent.

Quotation 5, pages 509–11. Under this heading the Oral Hygiene Committee questions the work of our dental hygienists and our means of determining their abilities. One of the sentences in their criticism reads: "There is no *evidence* of any particular establishment of ability in the 'observers' *uniformly* to detect the presence of pits and fissures and to classify them as directed and claimed." The Oral Hygiene Committee is mistaken. The Committee evidently overlooks the full significance of a correlation of .90 representing the objectivity

of examiners. It is clearly stated in the Monograph on page 43. On this page also is a brief description of the nature of the tooth examination. A footnote on this page reads: "Six dental hygienists and a dentist were employed in the preliminary weeks of experimentation to obtain adequate training and objectivity in the distinctions. The data were finally gathered by two dental hygienists." Another footnote on this page reads: "The method of recording and further details as to the directions followed in the examinations are reported in Appendix A." Appendix A occupies pages 71 to 79 in the Monograph.

In addition to the above there is another matter that the Oral Hygiene Committee should have mentioned in this connection. The dental hygienists making the examinations of enamel defects reported in Monograph III were trained to follow the technique outlined by the Hyatt Study Club, a copy of this technique having been presented by Dr. Hyatt to the medical director of our School Health Study Staff. Considerable time was given to discussing this technique with Dr. Hyatt, [12] Dr. M. Diamond of the Columbia University Dental School, and Dr. Dwyer,[12] then Dr. Hyatt's associate. The relative advantages of different types of explorers, the use of bifocal lens, the importance of carefully cleaning all the grooves with the explorer, wiping out with cotton, drying with alcohol, using the air syringe for the removal of saliva and the practicability of actually measuring the different sizes of enamel faults, were discussed with Dr. Hyatt and Dr. Diamond. On page 510, the Oral Hygiene Committee states in connection with the method used to establish the ability of our examiners: "The reader is given no opportunity to evaluate the means employed." But the reader is given the means on the pages of the Monograph indicated above. Furthermore, Dr. Hyatt and Dr. Dwyer, with whom we were in close touch during this work, were asked to check the findings in one hundred children that our dental hygienists had examined, in order to evaluate the accuracy of the work of our dental hygienists. Dr. Hyatt stated that he could not take the time, but that he would send his assistant Dr. Dwyer. Dr. Dwyer examined several children that had been examined independently by both our dental hygienists and we compared his findings with those of the

[12] Both Dr. Hyatt and Dr. Dwyer were members of the sub-committee of five that prepared the protest for the approval of the Oral Hygiene Committee.

dental hygienists. Dr. Dwyer appeared satisfied that our two dental hygienists were making satisfactory examinations and would obtain findings very much the same as a dentist would. But because Dr. Hyatt at one stage had seemed to feel that we should have employed dentists to make these observations, we urged Dr. Dwyer to examine one hundred children so that the evaluation might be unquestioned by Dr. Hyatt. Dr. Dwyer assured us that it was unnecessary as he was satisfied as to the accuracy of the examinations of the dental hygienists.

When the analysis of the work of our dental hygienists was completed, and Dr. Hyatt questioned these results because they did not support his own views regarding prophylactic odontotomy, again we strongly urged that he should himself check the findings of our dental hygienists on one hundred children. This was not done.

The balance of the Oral Hygiene Committee's protest under this heading points out that our method of detecting stain was faulty and that inasmuch as our records on stain, tartar and gingivitis were admittedly not as dependable as our measures of caries, we should not have reported upon them at all. This is a curious point of view on the part of the Oral Hygiene Committee. On page 519 of the protest we are accused of concealing data and of presenting only selected portions. On page 511, however, fault is found with us for publishing some of our data because of its admitted and demonstrated imperfections. Let us refer to Chapter V of Monograph III. The title is "Stain, tartar and gingivitis." The opening paragraph reads as follows: "Measurements made of stain, tartar, and conditions of gingiva *for lack of a better method* were made by the use of rating scales in the hands of dental hygienists. Ratings are always less satisfactory than other types of measurement. Our estimates of decay are much better since they are spatial and so depend upon tangible sensory experience. These other estimates, however, consist of opinions regarding the severity of conditions and have no direct evidential verification . . . ." (Italic not in the original.)

Throughout this chapter we tried to give a plain statement of fact, of what we did, of the result of our analysis and an interpretation of this result. The fact that reliabilities of examiners were not high is not a valid reason for suppressing this information. We gave the

mathematical expression of the objectivities of each of the three examiners. The Appendix shows the method of recording these items. We pointed out the limitations of the data. In the summary we wrote: "Stain *as measured* is unrelated to tartar, gingivitis, or uncorrected caries" (italic not in the original). We fail to understand the Oral Hygiene Committee's point of view that if a result does not turn out as reliable as we should like it to be, then reference to such material should be omitted.

Quotation 6, pages 511 and 512. The Oral Hygiene Committee here tells us that we are all mixed up in our classification of the types of pits and fissures. They quote us as writing: "Type 3 (pit or fissure) may be considered as caries." Since we were investigating the practicability of detecting the dangerous enamel defects we wanted to be sure if possible to find the differences in enamel faults that would distinguish those teeth most likely to decay. We, therefore, consulted Dr. M. Diamond as a dental anatomist to be sure we understood the anatomical considerations involved. We discussed with Dr. Hyatt the problem of differentiating the depth of the pits and fissures. We experimented at length and found after examining many teeth that (apart from the difference between pits and fissures) there were three main distinctions in each which our dental hygienists could make accurately. These we arbitrarily called Types 1, 2, 3. All through these experimental examinations we had each child examined more than once by two or more examiners. The fact that objectivity between them was demonstrated indicates that they were not confused in distinguishing these types. They were able to make like distinctions when working on the same children independently.

In some enamel defects it was apparent that the explorer wedged or caught between the walls of enamel on each side of the opening. This we called Type 1. Other pits and fissures were more open and the explorer did not wedge in the opening. In this latter group of enamel defects it was found that the consistency of the enamel at the base and on the walls varied in resistance to the sharp explorer. Those openings with firm, hard, resistant base and walls were arbitrarily classified as Type 2; those less resistant or with "softening of the base or walls of the enamel" were classified arbitrarily as Type 3, while possibly bordering on caries. We therefore treated the data for each

type separately so that conclusions may be formed if they are considered as enamel defects or if they are considered as caries. Regardless of the decision the conclusions are identical. This is the explanation of our expression, "Type 3 (pit or fissure) *may* be considered as caries" (italic not in the original). The Oral Hygiene Committee is mistaken in trying to read into this section a contradiction that is not there. The reader is not apt to have difficulty if the entire text is before him. He most probably will be confused if presented merely with unqualified isolated selections from a scientific text.

On page 45 of the Monograph we use the phrase, "protective influence of Types 1 and 2." The Oral Hygiene Committee ridicules "protective." Its use in the above sense is the language of the biometrician. Protection can be both positive and negative. Ordinarily a negative protection would be termed a hazard or a danger. Take the matter of resistance to pressure. Some things we call hard, others soft. The biometrician would be inclined to speak of the entire range of this quality in terms of degree of hardness. A very soft substance would have a very slight degree of hardness. Similarly the author of Monograph III spoke of the "protective influence of Types 1 and 2" pits and fissures, even though this might be a negative protection or a hazard. This is an instance of a lack of a common language resulting in a misunderstanding.

Quotation 7, pages 512–14. In these three pages the Oral Hygiene Committee states that we are unfamiliar with established conclusions of modern dental research. We are told that "the correlation of decay in one tooth with decay in another tooth is not a relation of cause and effect." The Monograph had not stated that this was a cause and effect relationship. Without submitting any evidence or references the Oral Hygiene Committee presents its viewpoint on the relationship of decay in teeth and the liability to caries that is shared by different teeth. Examples of these didactic opinions follow (page 513): "Bilaterality of liability is common to all teeth in horizontally opposite situations . . . . . When the conformation or structure of the enamel at a particular location on a tooth is such as to afford undisturbed retention of bacteria, for example, there is very frequently, in fact almost always, the same conformation or structure of the enamel in the similar location on the tooth horizontally opposite." It would

have been extremely helpful if reference to research supporting these statements as well as others on the same pages had been given.

The Committee seems to be so impressed with the idea that a preventive operative procedure is the panacea for the dental ills of children that it colors most of the Committee's criticism. Whether relevant or irrelevant they have repeatedly introduced it. A striking example is their comment on page 513: "The degree to which filled or unfilled caries in one tooth is associated with filled or unfilled caries in another tooth does not and cannot measure the *effect* of the *influences* of decay, but when all surfaces and all teeth are considered, *the possibilities of an operative preventive procedure may be determined.*" (Italic in last clause not in the original.)

Quotation 8, pages 514–520. Six pages of comment are called forth by the following quotation from the Monograph (page 48): "The fact that such enamel liabilities as do occur, so frequently remain non-carious up to the age of twelve, is a strong reminder that there is such a thing as organic immunity which is not yet understood." We call attention to the similarity of this thought of ours and the quotation from Dr. Johnson given above. And still the Oral Hygiene Committee calls this reference of ours to immunity "irrelevant" and "mystic." Susceptibility and immunity as discussed by Dr. Johnson certainly deserve consideration as an explanation of why so many enamel defects remain non-carious up to the age of twelve. Furthermore in the above mentioned article Dr. Johnson writes:

> "I have never seen any reference whatever by the advocates of this practice to the question of susceptibility or immunity as a factor in deciding whether or not a fissure shall be drilled out and filled."

If the Oral Hygiene Committee should admit that any less than a great majority of the enamel defects are susceptible to decay, or that immunity is important, then they would recognize at once the import of our findings and our interpretations. They would then see why we contend that a method of distinguishing pre-carious enamel defects is much needed, and that a public health or clinic policy of filling non-carious pits and fissures as it is now understood is not the best kind of prophylactic odontotomy.

Our statement was made as a result of an analysis of records of

enamel defects in non-carious first molars in children six to thirteen years of age, which indicated conclusively that the proportion of non-carious teeth that have no enamel defect did not increase in the older age groups as would be expected if a large majority of the enamel defects found do predispose to caries. Furthermore, the number of enamel defects of any one of three types found per non-carious tooth remained the same throughout the age groups. The fact that under some classifications Type 3 pit and fissure might be considered a beginning carious cavity (see reply to Quotation 6) does not alter the case as the analysis is presented separately for each type.

The Oral Hygiene Committee has made three main criticisms of this analysis and conclusion. The first of these on page 515 of the protest is that the data did not allude "to the *same children at successive periods of observation* from age six to age thirteen, nor to *all* of the first molars in the children examined, but instead to different children examined once individually, and only to the so-called 'healthy' or 'non-carious' first molars remaining in a few of these children at the time of examination." The criticism relative to examinations of the same children at successive periods might be applied equally well to all the existing data on the problem, including that which is presented in the Oral Hygiene Committee's protest (the Appendix excepted) and we have found no published record of clinical experience which includes repeated examinations at successive periods. We would welcome data of the kind mentioned if they were objectively obtained. With reference to the Committee's criticism that our data were limited to "healthy" or "non-carious" teeth, it is highly irrelevant to include carious teeth in an investigation of the relative proportion of non-carious teeth with enamel defect. All the non-carious first molars in the children examined were included in the analysis.

The second criticism alleges that the author of Monograph III was not open minded in the treatment of the data, as he ignored the suggestion of progression from Type 1 pit or fissure, to Type 2, thence to Type 3 and finally to caries. The argument of the Committee follows (page 515): "If, however, for *temporary* purposes of analysis, we *assume* with the author that each average in Table 20 (page 45) for enamel defects is truly representative of conditions in the corresponding age-groups, then we note also that the number of defects increases

in sequence from Type 1 to Type 3 not only *horizontally*, for a given age as he stresses them, but also when followed diagonally downward to the right *in time* for *successive ages, as shown in Table 2*, page 517, of the present report. These relationships should have *suggested*, to an open-minded investigator, the *possibility* that, at the time the enamel defects were found, some unobserved defects were enlarging toward Type 1; some of Type 1 were becoming Type 2; and some of Type 2 were approaching Type 3. Nothing in Monograph III shows that this *possibility* was actually tested as a mathematical alternative, the author having apparently made no effort, from a study of a given group of children, to *exclude* it. He did not ascertain whether *any* enamel defects, *undetected by his procedure*, became at each successive recorded age *gross enough for detection by these means*. He also failed to learn whether, at the other extreme, an approximately equal number of defects of Type 3 became so obviously carious that the teeth containing them were *excluded from the so-called 'healthy' group*. This is a striking double omission from a plan for a supposedly unprejudiced study."

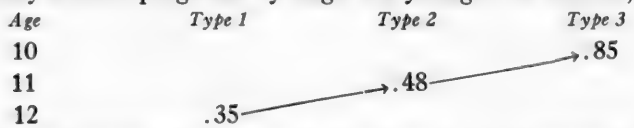
For the purpose of this argument the Committee added together the figures for pits and fissures by type for each age and presented them in Table 1 (page 516). The figures are here reproduced to indicate their reasoning.

Average total number of each type of enamel defect per "healthy" first molar

Age	*Type 1*	*Type 2*	*Type 3*
6	*0.22*	0.41	0.76
7	.19	*.45*	.68
8	.24	.35	*.66*
9	.23	.50	.64
10	.28	.51	**.85**
11	.28	**.48**	.78
12	**.35**	.50	.67
13	.19	.39	.64

We have italicized the figures for Type 1 at six years of age, Type 2 at seven years of age, and Type 3 at eight years of age; have connected them with arrows pointing downward; and in that way find the progression referred to by the Committee. The same relation is also true

for all other parallel sets of figures. This indicates according to the Oral Hygiene Committee, that one type of pit and fissure progresses on to the next type in the course of time. But the Oral Hygiene Committee is playing with the figures. We give some of the figures in bold-faced type and have connected them with arrows pointing upward. Instead of going from younger to older ages, the lower arrows proceed backward from the older to the younger ages. By this process, and using the reasoning of the Oral Hygiene Committee in the cause of which we are berated for our lack of openmindedness, we may say that instead of pits and fissures becoming progressively larger the older the child is, they really become progressively larger the younger the child is, thus:



The Oral Hygiene Committee is mistaken in its interpretation of this table. There are more Type 3 pits and fissures at all ages (between six and thirteen) than there are Type 2, and more Type 2 at all ages than Type 1, hence a larger occurrence can be shown for Type 3 relative to Type 2 and for Type 2 relative to Type 1 no matter what order of age groups are chosen. This increase, however, has no bearing whatever upon progression since there is no significant difference in proportional occurrence between ages.

The argument of the Committee incidentally is contrary to the judgment of Dr. Hyatt at one time, for on December 10, 1929, he wrote:

"As pit or fissure caries do not increase at the surface, therefore, there is no reason that type 2 would become type 3."

Furthermore with such data as were presented in the foregoing table, such a progression as suggested by the Oral Hygiene Committee could not take place unless there were undetected pits and fissures that later progressed to Type 1. The Committee says in the protest that "the author of Monograph III *sensed* the probability that there were enamel defects of the undetected, and therefore of the *uncounted*, kind in his work." (The word "sensed" not italicized in the original.) The author not only "sensed" that there were undetected pits and fissures but called attention to the fact on page 42 (Monograph): "It

may very well be that the most dangerous types of enamel defect are those that cannot be observed until the groove is cut into with a burr."

In any public dental health examination made at a public clinic, there would very likely be many pits and fissures so small that they *would not be detected* and these are just as likely to become caries as those that *are detected*. Since they are not discovered they would not receive the alleged benefits of prophylactic odontotomy were such a program in force. It is important therefore to find some means of identifying all pits and fissures that are likely to become carious before it may be considered as proper policy to advise prophylactic odontotomy in public clinics. And this is the main point which we believe the Oral Hygiene Committee failed to recognize.

As the third criticism, we are accused of withholding data which would not support our contention. The table which the Committee

TABLE 1

Comparative data from page 519 of the protest

AGE	DATA IN VARIOUS PARTS OF THE MONOGRAPH ON NON-CARIOUS FIRT MOLARS		DATA IN TABLES 18, 19 AND 20 OF THE MONOGRAPH, ON ENAMEL DEFECTS IN NON-CARIOUS FIRST MOLARS	
	Total number of children	Total number of non-carious first molars	Total number of children	Total number of non-carious first molars
10	1,157	1,664	157	438
11	1,636	1,923	179	475
12	1,149	1,193	138	319
Total.....	3,942	4,780	474	1,232

has constructed (page 519) to convict us of this error is here given as *Table 1*. It is to be noted that data are given for 3942 children in the left side of the table, and for only 474 children in the right side of the table, which deals with enamel defects, the subject discussed in Chapter IV of the Monograph. Why did we select only about 12 per cent of the children and only about 25 per cent of the non-carious first molars to support our conclusions in Chapter IV regarding enamel defects and their relation to decay? The protest on page 519 proceeds as follows: "The alert reader can only speculate on the hidden reasons for these surprising omissions of most of the relevant data. Was this

gross departure from the scientific method due to accident, or to carelessness; or did the excluded data prove something contrary to predetermined conclusions. . . . . The facts in this paragraph of our comment do not support a belief that the Monograph is an accurate and sincere presentation." When the Oral Hygiene Committee discovered this apparent discrepancy, two possible explanations might have occurred to them. One was that we did not possess the data on pits and fissures of the missing children; the other, that we were in possession of the figures but voluntarily concealed them. The Committee chose the latter conclusion.

Even if the Committee as a whole did not know the actual facts, some of its members were obviously aware of them. In the Foreword of the Monograph (page vii) we had written (italic not in the original): "The source of the data for these publications is field measurement of about 7500 public school children of the *fifth* and *sixth* grades . . . [age groups 10 to 12 inclusive] . . . . . Supplementary data were also obtained from other groups of children." In Chapter IV of the Monograph, on enamel defects, we wrote (page 41): "In order to study the importance of this aspect of dental care we examined non-carious first molars in the mouths of children varying in age from *six* to *thirteen*" [school grades 1st to 8th]. (Italic not in the original.) In the Appendix, on page 539 of the Oral Hygiene Committee's protest, is this statement as a footnote: "In the first paragraph of the Foreword of the Monograph, it is stated that the data were obtained 'during the school year 1927–28,' and that 'supplementary data were also obtained from other groups of children.' The data of the first examination in this table were evidently among the supplementary findings." Further than this, a statistician associated professionally with Dr. Hyatt, at his request called the author of the Monograph on the telephone and asked about this discrepancy in numbers. *He was told that data on pits and fissures were not recorded for the 7500 children of the main study,* but were limited to about 1000 children of ages 6 to 13 in the supplementary study.

The actual number of cases that were examined for pits and fissures was 1199, but in 473 of these children none of the first molars present in the mouth were non-carious, hence it was impossible to include them in a study of non-carious teeth. Of course, some of the first molars

were unerupted; others had been extracted. In view of the above opportunities for the Committee to know that two entirely different sets of data were treated, one of which did not include records of pits and fissures, we wonder why such an unwarranted criticism was ever made.

Quotation 9, pages 520–23. On page 42 of the Monograph, a table (18) was presented showing the per cent of non-carious molars which are free from pits and fissures at various ages. This is repeated below.

Age	Number of non-carious teeth examined	Percent of these teeth *free* from enamel defect
6	238	34
7	371	32
8	354	34
9	364	27
10	438	16
11	475	20
12	319	22
13	173	25

If *recognizable* pits and fissures predispose to decay we should expect that the proportion of non-carious molars free from recognizable pits and fissures would increase with age, because the molars with detected pits and fissures would have proceeded on to decay leaving as survivors in the older age groups the teeth without such pits and fissures. The above table shows no such increase. In fact the proportion of non-carious molars free from recognizable pits and fissures is less at the older ages than at the younger ages within the range of 6 to 13 years. It is this fact which gave rise to the statement appearing in the Monograph (page 41) that "the teeth that escape caries are more apt to be those that have remained healthy in spite of having observable enamel defect."

The Oral Hygiene Committee criticises the data here by first presenting the data of the above table, using an inaccurate graph that does not pass through the points that should be represented. They thus present an optical illusion which tends to show that the rise in percentage from ages 10 to 13 is significant. Graphs should never be used for this purpose since the choice of coördinates may conceal or accentuate any chance variation. The significance of the difference

between two percentages is not decided by means of graphs, but by strict statistical means. It may be shown by recognized statistical procedures that the difference that occurs between the percentage at 10 years of age and the percentage at 13 years of age is not greater than might occur by chance. Such a determination would have been far more valuable to the Committee had they really desired to know the significance of these differences. The Committee continues to confuse the data by performing computations on selected age groups to prove its point. It excuses this procedure by stating that these were the three age groups on which we had the most adequate data in the main body of the study. This is of course entirely irrelevant for, as explained in our reply to Quotation 8, the data on pits and fissures are entirely distinct from the data otherwise treated in the Monograph.

In a table on page 522 of the protest, the Committee calls attention to the fact that with the advance in age, the percentage of defective non-carious teeth decreases in the same amount that the percentage of unhealthy teeth increases. The argument is then advanced that the unhealthy teeth must come from the defective teeth. (See *Table 2.*)

TABLE 2

Distribution of permanent first molars, per hundred of possible maximum number

AGE	CHILDREN	NON-CARIOUS					UNHEALTHY (CARIOUS) OR MISSING
		Total	Defective		Free from defects		
yr.	*no.*	*no.*	*no.*	*per cent*	*no.*	*per cent*	*no.*
10	157	69.7	58.5	(84)	11.2	(16)	30.3
11	179	66.3	53.0	(80)	13.3	(20)	33.7
12	138	57.8	45.1	(78)	12.7	(22)	42.2
1	2	3	4	5	6	7	8

Of course, this is unsound reasoning, but in *Table 3*, where we have reproduced the entire data, we see that the Committee was aided by a chance deviation in the percentage of defective teeth at age ten. Were we to use the same incorrect reasoning as used by the Committee we would conclude that for ages prior to ten the unhealthy teeth came from the teeth that were free from enamel defects. Of course such specious reasoning with selected age groups of the data is unwarranted.

TABLE 3

Distribution of permanent first molars, per hundred of possible maximum number

AGE	CHILDREN	NON-CARIOUS					UNHEALTHY (CARIOUS) OR MISSING
		Total	Defective		Free from defects		
yr.	*no.*	*no.*	*no*	*per cent*	*no.*	*per cent*	*no.*
6	76	78.3	51.7	66.0	26.6	34.0	21.7
7	117	79.3	53.9	68.0	25.4	32.0	20.7
8	116	76.3	50.4	66.1	25.9	33.9	23.7
9	121	75.2	54.9	73.0	20.3	27.0	24.8
10	157	69.7	58.5	83.9	11.2	16.1	30.3
11	179	66.3	53.0	79.9	13.3	20.1	33.7
12	138	57.8	45.1	78.0	12.7	22.0	42.2
13	74	58.4	43.8	75.0	14.6	25.0	41.6
1	2	3	4	5	6	7	8

The Committee then points out three limitations of the research described in the Monograph. They are: (a) Observations on a given group of children were not made recurrently during a succession of years to ascertain whether enamel defects that existed in healthy first molars in the earlier years, but were not detected, became carious in later years. (b) The possibility was not excluded that enamel defects in first molars at ages from 6 to 13 develop or get large enough in these teeth thereafter to become detectable by the means employed. (c) The age at which the observed enamel defects in the first molars of the older children were first detectable was not indicated. The first of these has been answered under remarks on Quotation 8. The second and third are problems for future research, but are irrelevant to the data necessary for an answer to the question to which we addressed ourselves. We repeat that this question is the ability to discriminate under public clinic conditions between enamel defects that are dangerous and those that are not. There are many other problems that still demand research. We wish we could induce the Oral Hygiene Committee to engage in more substantial research so that it could combat our findings with scientific evidence instead of professional mandates and such statements as "every dentist knows."

The Oral Hygiene Committee further states (page 523): "It is well known that in the older age groups, the *proportions* of non-carious teeth having no observable enamel defect increase with age because

most of the teeth having enamel defects become carious." The Committee then presents a table for age groups 6, 10, 13, 14–16, 18–30, 30–40, and states that the data for age groups above 14 were available before the beginning of the study described in Monograph III. Judging from our conversations with Dr. Hyatt, the above data were not available. Data showing the proportion of surviving non-carious first molars with and without enamel defects were available for the age group 30 to 40. If we are not mistaken, data for ages 14–16 and 18–30 were either gathered or worked up while Monograph III was in preparation. The table containing these figures presented by the Committee includes no reference to the source of the data for ages 14–16, 18–30, and 30–40 in order that the manner of gathering the data, the objectivity of the examiners, the type of groups sampled and the number in the sample may be known. All of these latter are pertinent facts.

Comment on this table, at the bottom of page 523 of the protest, establishes the Committee's complete comprehension of the manner of handling a single (in distinction from repeated) examination of different age groups as reported in the dental Monograph. This may be shown by contrasting the following two statements, both in the protest. The first one shows an intelligent comprehension of the method used by us. The other one sounds as if the authors had entirely failed to grasp the significance of the same method. In the first quotation the assertion is made in favor of the Committee's contention. In the other one the Committee claims to be confused by the very same logic but stated by us as unfavorable to the Committee's contentions. If increase in mortality with age argues for given preventive measures, then a constant mortality as age increases must argue against it. The statements in the protest are these (italic not in the original):

"This summary also indicates that owing chiefly to lack of preventive measures, which are attainable in the main only in childhood, *the mortality of non-carious first molars having enamel defects increases cumulatively with age.*" (Page 523).

"There is no evidence in the Monograph of any recurrent examination of the same children and teeth to justify these conclusions, which in the main *appear to indicate predictions* rather than observed conditions." (Page 507.)

Quotation 10, pages 523–25. Under this quotation the Committee criticizes this statistical finding on page 43 of our Monograph: "A tooth without such defects as we are able to observe is just as likely to become carious as are the others." First it unjustly misrepresents this conclusion by picking three statements out of context and quoting them together. Forced to stand independent of the subject matter and through the Committee's use of italics they are made to appear as contradictory. The statements are:

"The teeth that *escape caries* are *more apt* to be those that have remained healthy in spite of *having observable enamel defect* (italic not in the original)."

"A tooth *without such defects* as we are able to observe is *just as likely* to *become carious* as are the others (italic not in the original)."

"First molars which seemed to our observers to have *no enamel defects* were *more liable to caries* than were those which could be described by them in terms of pit and fissure (italic not in the original)."

The first and third statement are identical in meaning. The second statement, because of the subject matter immediately preceding it in the context of the Monograph, is simply a conservative reiteration of the other two statements. Hence the juggling of the statements and implied contradictions are without justification.

The second criticism of this finding is aimed at the reliability of the data. In this regard it should be noted that the number of children studied in each age group is small enough to permit deviations for each age, yet the number is still sufficient to give reliable results—reliability being understood in the biometric interpretation that any random half of the records give the same conclusion as any other random half. Of course reliability is a statistical question and the protest gives evidence throughout of failure either to procure or to heed competent statistical guidance.

Quotation 11, pages 525–27. In discussing the differentiation of enamel defects by types the author of Monograph III wrote as follows (page 44 of Monograph III): "It is obvious that carious teeth should not be considered in these observations of enamel defect, because the pits and fissures which have been involved in the caries cannot be known. Teeth which escape caries may, however, be studied in regard to enamel defect. As children grow older fewer first molars sur-

vive and those that do should show more plainly any characteristics that protect against decay. Because enamel defects and caries occur most frequently on the occlusal surfaces of first molars we have limited this study to these areas." The protest quotes the first sentence in the above and comments as follows (page 525–526): "On the contrary it is *obvious* that the prevalent locations of caries in carious teeth, and also of non-carious surfaces on carious teeth, should not have been disregarded. Why? Because caries in comparable adult teeth usually occurs in *similar locations*, which include all surfaces where enamel defects are present, and in the *same places* on these surfaces."

The Oral Hygiene Committee misunderstands the meaning of the Monograph quotation. Carious teeth cannot be considered in this portion of the discussion of enamel defects because it is impossible to judge whether there were or were not observable pits and fissures in these teeth prior to caries. The Committee implies that, because caries occurs most frequently on the same surface of the tooth in which most observable pits and fissures occur, therefore observable pits and fissures are responsible for caries. Bacteriologists do not conclude that, because certain bacteria are present when a disease is present, and are absent when a disease is absent, those bacteria are therefore the cause of the disease. So with dental caries: a much more rigid proof is needed before we can accept the prognosis from observable pits and fissures put forward by the Committee. For instance, if our main contention is correct that enamel defects which escape detection are dangerous, then these too occur most frequently on these same surfaces. The liability of certain surfaces to both enamel defect and caries may be due to some common cause, and does not necessarily imply a causal relationship one to another.

The Committee presents a table on page 528 with data from Monograph III and elsewhere (source of other references not given) showing numbers of healthy and unhealthy molars by age. The attempt is made to conclude from this that because the first permanent molars tend to decay, something should be done to prevent this destruction. Agreed; but how can the decay be prevented? Will the filling of observable pits and fissures best insure teeth against further decay? That is the question before us. The problem is to find methods of distinguishing between teeth that will decay and those that will not.

Quotation 12, pages 527–29. The Oral Hygiene Committee presents the following point: "In the general application of preventive measures, mortality is a decisive factor. When the mortality is high, it is desirable to apply a preventive procedure to all, regardless of the probable immunity of some." Prevention is the ultimate goal toward which we are all striving. But is the Oral Hygiene Committee satisfied that a true preventive measure is at hand? Is there available a saisfactory technique for the identification of dangerous enamel faults? Would it be practical to use present methods in public clinics? Dr. Hyatt once wrote in a letter to a member of our staff (June 17, 1930): "First it must be borne in mind that no operative preventive dental procedure has as yet been carried out on any plan which would warrant deductions being made for or against the procedure. There have been some isolated cases which are full of promise. They are not numerous enough, however, to warrant any dogmatic assertion."

Quotation 13, page 530. On page 19 of the Monograph is this statement: "The spread of caries is not due to any localized cause but to some influence common to all parts of the mouth." This quotation, isolated from the two explanatory sentences that followed it in the Monograph, is made the subject of criticism in the protest. If the two other sentences had been understood there would have been no difficulty. These two sentences are: "Being in the mouth at the same time when a tooth decays and so being subject to a common influence is the determining factor in the correlated decay of another tooth. It is also evident that the influence does not act with equal force throughout childhood." As far as our data are concerned this generalized influence may either be present in the mouth or may be the result of some metabolic process. The data indicate that all occlusal surfaces are subject to this generalized factor when it is active. Therefore the reference to surfaces in the Committee's document has no bearing on the issue.

Quotation 14, pages 530–32, from the Monograph, is here repeated: "Were caries the product of accidental combinations of circumstances then there would be no real correlation between decay in any given tooth and the decay in another. Then we would have obtained a chance distribution of r's with zero as their average. Since all correlations are positive we must assume that either

the caries in one tooth affect [sic] the caries in another, or that caries are [sic] in part a common result of some influence which operates on both teeth. We have noted in Chapter I that these positive correlations vary in size. This means that the influence that teeth have upon each other, or the degree to which teeth share some common influence, varies with the type and position of the teeth concerned (page 13)." The Oral Hygiene Committee here selects most of the introductory paragraph of Chapter II of our Monograph for criticism. The Committee evidently has not appreciated the meaning of a correlation from its statement that "the author decides, from mathematical calculations alone, that 'accidental combinations of circumstances' cannot be factors in any decay." The author does not decide this. This statement is made on no mathematical calculations. He means: if there is a correlation between decay in one tooth with decay in another tooth, such a correlation could not happen as an accidental combination of circumstances. It must occur because of some influence common to the teeth which are found to be related, or, of course such correlations might occur as a result of caries in one tooth affecting caries in another tooth.

We might discuss here just how such factors as mechanical injury, destructive tooth-cleaning fluids, systemic diseases, inadequate diets, etc., might be interpreted as having a bearing upon the data presented. However, as the Committee appears to be most interested in discussing structure and conformation of enamel surfaces because of its bearing on the support of prophylactic odontotomy, we shall merely call attention to the fact that the quotation above and Chapter II are concerned with interrelationships of the teeth and there has been *no disregard of the effect of local conditions*. The data show on the other hand how the effects of local conditions are less important than the influences which are common to teeth in different parts of the mouth. We might suggest that these findings fit in very well with the concepts of the experimental workers concerned with the study of diet in relation to caries.

It is interesting how the protest leads the comments directly into the familiar subject of variations in surface conformation and variations of enamel structure, and asserts caries "is a local disorder." The Committee then refers again to the argument for prophylactic odontotomy as follows (page 532): "Studies of the teeth of adults have indicated

the surfaces, and the specific locations on each surface, where dental decay commonly occurs early in life. These data suggest the nature, extent, and applicability of a practical preventive procedure." The Committee cannot refrain from again asserting that "Monograph III has not shown that these current views in dentistry are unfounded." We agree. Monograph III had no intention of treating this subject. We have discussed the adequacy of the logic above.

Quotation 15, pages 532-33. The Oral Hygiene Committee endeavors to show contradictory statements in our Monograph by picking sentences out of their context and placing them side by side. There is no contradiction if the reader follows the logic in the sequence presented in the Monograph. The Monograph had in all seriousness made reference to "waves of influence" to distinguish from influences on decay that were continuous. The Oral Hygiene Committee tosses this "waves of influence" into the air like a kitten with a mouse and enjoys the antics hugely. The Committee makes this statement: . . . . "then the act of *'filling the cavities'* so completely and mathematically frustrates the 'waves of influence' that they immediately cease to wave." We confess we had a broad smile ourselves over this page of the protest.

General remarks (pages 534 and 535)

On pages 534–35 the Oral Hygiene Committee sums up its case, which consists of a series of condemnatory statements. We have already covered in detail the points raised in this summary. The last paragraph we quote as a good example of the Oral Hygiene Committee's reasoning through its 50-page protest: "Results beneficial to the health and well-being of school children, improvement in their deportment, and reduction of retardation in their school progress with less waste of the taxpayer's financial contribution to public welfare, as assured consequences of an effective oral-health policy in schools, have apparently not interested the American Child Health Association. The Monograph totally ignores these well established facts, although there are thousands of authentic instances of such results of dental health service for children in public schools." We are not sure that we understand this statement. Does the Committee mean that some of the above accomplishments have resulted from a policy of

filling enamel defects? Evidently not because on page 534 of the protest the Committee writes: "Mass procedure in operative preventive work in public dental-health programs has hardly been begun."

Does the Committee mean that much good has come from the dental health services established by schools, meaning reparative work, the encouragement of mouth cleanliness and instruction in diet? If this is what the Committee means then we agree. We too believe that this type of work has done much good. However, this must not be taken to mean that we approve of such unqualified statements as the one quoted above from the protest.

If the Committee means to imply that the American Child Health Association is not interested in a dental health program in the schools, the Committee is mistaken. This is a matter in which the American Child Health Association is intensely interested, so interested in fact that in the School Health Study it included measurements of mouth conditions and observations of the administration of dental service in the schools. The American Child Health Association is so interested that it has conducted a symposium on dental health education at its annual meeting. It has contributed to the promotion of care of children's teeth by dentists through national, state and local programs. The staff of the Association is informed on the literature of both the dental and public health field relating to preventive dentistry and public health dental policies. The members of the staff have sought to inform themselves through surveys, contacts with the American Dental Association officials and many dentists concerned with preventive dentistry so that they can use the numerous opportunities which are presented to pass these ideas on to communities all over the country. In spite of its interest the Association has not been able to find the evidence that an effective oral health policy has done through public clinics those things which the Committee tells us are assured.

Our Monograph was devoted to a study of a limited phase of this matter; namely, how to secure a comparative measure of mouth conditions, the factors identified with decay, and the possibilities of a policy of filling observable pits and fissures in public clinics as a means of preventing decay. There is nothing in Monograph III which would warrant the Oral Hygiene Committee in concluding that the American

Child Health Association was not interested in "an effective oral health policy in schools." There is nothing in the Monograph that would warrant the Committee in stating as it did in the abstract of the protest, page 499: "A do-nothing policy, as affecting dentistry, is a logical implication from his conclusions," referring to the author of Monograph III.

Recommendations as to procedure (page 535)

The Oral Hygiene Committee here recommends (on page 535) that "(a) a thorough study of Monograph III be undertaken by the Oral Hygiene Committee of Greater New York, with the coöperation of leading dental organizations and other advisers; and that, at the conclusion of the study, (b) the merits of the Monograph be set forth, and (c) its deficiencies, unwarranted deductions, and misleading statements be promptly noted and corrected—for the information of the general public," etc. Isn't the Committee, in recommendation (c) mildly suggesting what the outcome of the study should be? Wouldn't it be better to let the deficiencies evolve voluntarily? However, waiving this point, we can express our very great satisfaction with these recommendations. We only wish that a thorough study of Monograph III had been made *before* the protest was written and published. Then perhaps there would have been less misunderstanding, less irrelevant matter, and less emotional display in the protest. One addition which we should like to see made to these recommendations is that statements disputing the interpretations made in the Monograph be accompanied by evidence.

Appendix (pages 536–46)

An eleven page Appendix is attached to the protest in which some of our original data are presented together with records of reëxaminations made thirteen months later by one of the members of the Oral Hygiene Committee. Several criticisms which appear in the main body of the protest are also repeated here. It is of course unnecessary to repeat the answers to these criticisms. The data are summarized in several tables, none of which were made by the method described in the Monograph. When we were studying the question, Are teeth that have *observable* enamel defects more likely to decay than teeth free

from such faults?, all of the non-carious teeth were used *irrespective* of whether the first molars were in place or not. The Committee, however, used only children in which all first permanent molars are in place. When the tables appearing in this Appendix are constructed using all the non-carious teeth, the discrepancy between the data in the Monograph and the partial data thus quoted is much less than the discrepancy shown by the Committee, and the data appear much more consistent from age group to age group.

Data on 115 non-carious molars are certainly not sufficient to determine the relative liability of defective and defect-free molars to decay. Assuming that prophylactic odontotomy does prevent future decay, Summary A on page 542 shows *that 44 of these 115 teeth would not have received the advantages of treatment in a public clinic because they would have appeared free from defects. Yet 21 of these were affected by caries in 13 months.* In fact this is strong corroboration of our original points. It indicates both frequent occurrence of teeth that have no observable enamel defects, and large liability of these teeth to caries.

Therefore it would have been much more profitable if the Oral Hygiene Committee had concerned itself with the problem of identifying those teeth susceptible to decay in the interests of a preventive procedure instead of firing a broadside criticism at a research that was intended to clear the way for just such work.

370 Seventh Avenue

DENTAL DISEASE[1]

Notes on Its Prevention

Communicated by J. MENZIES CAMPBELL, D.D.S., L.D.S., F.R.S.E.

Glasgow, Scotland

We are impressed by the great amount of suffering and ill-health which is caused by dental disease. Believing that most of this could and should be prevented, we feel it desirable to outline, for the benefit of the public, the means, which, we consider, would conduce to that end.

Diet. Correct diet should begin before birth, i.e., the mother should be correctly fed during the nine months she is carrying the child and the months she is nursing it. Strong grounds exist for believing that many of the mal-developments of the body, including contraction of the jaws and consequent irregularity of the teeth and also most of the 'diseases of civilisation,' arise from an insufficiency of vitamins A, B, C, and D in the food, not only during development, but also throughout life. Correct feeding, together with all the other measures which go to improve general health, reacts favourably on the state of the mouth and tends to prevent dental disease.

The actual cause of decay of the teeth is external to the teeth themselves and consists of an acid, produced by the fermentation of starchy and sugary particles, which remain in the mouth and which are derived chiefly from white bread, confectionery, sugar and sweets. Raw fruit, raw vegetables (e.g., celery and salads) and nuts, all of which require mastication, are especially valuable, as, by their means, fermentable particles are cleared away and they themselves leave no fermentable residue. Fruit, e.g., apples, pears, oranges, is particularly valuable at the end of a meal, because, when masticated, it not only removes other food particles but it also causes an increased flow of saliva of an increased alkalinity—this neutralizes the acid. Potatoes are an excellent food and contain, like fruits and other vegetables, vitamins in moderate quantity; but should also be followed by fruit, as they are not innocent regarding fermentable particles.

Oral hygiene. In addition to the above natural means of cleansing,

[1] This manuscript has been sent to the British press.—[J. M. C.]

there remains the artificial aid of the tooth-brush. Brushing the teeth and gums preferably with salt (one teaspoonful) and water (one tumblerful) should be practised daily; when done only once daily, it should be after the last meal. The gums themselves must have active friction in every part, behind and before, to help to prevent sponginess and pyorrhoea. The teeth should be thoroughly brushed with saline water in every accessible part, both across and up and down.

Protessional help. In addition to all these measures the teeth and gums should be regularly inspected and, if necessary, treated by a dentist. A painful and neglected tooth may prevent thorough mastication and so lead to stagnant deposits, in the mouth, of germs and food particles and thus favour further decay.

Summary. To prevent dental decay (caries) and pyorrhoea: (1) Eat bread and other products made of wholemeal flour or of stone-ground white flour. (2) Eat foods containing all the vitamins. (3) Eat toast, crusts and other firm materials requiring mastication, in preference to soft, sticky foods. (*Note:* Vegetables, which require to be cooked, should be cooked for as short a time as possible.) (4) Eat raw fruits, nuts and raw vegetables, especially at the very end of a meal. (5) Brush the gums and teeth, without fail, after the last meal of the day or just before going to bed. (6) Visit a dentist, twice a year, for the inspection and, if necessary, treatment of the gums and teeth. The intelligent and steadfast practice of the means indicated in these rules would result in far fewer teeth being lost from dental decay or pyorrhoea.

Signed: (Sir) Harry Baldwin, C.V.O., M.R.C.S., L.D.S.,
Hon. Dental Surgeon to H. M. the King
(Sir) Norman Bennett, M.A., M.B., B.Ch., L.D.S.,
President, British Dental Association
J. H. Badcock, M.R.C.S., L.D.S.,
Ex-president, British Dental Association
William Guy, LL.D., F.R.C.S., F.R.S.E., L.D.S.,
Dean, Edinburgh Dental Hospital
William Hern, O.B.E., M.R.C.S., L.D.S.,
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J. Menzies Campbell, D.D.S., L.D.S., F.R.S.E.,
Member, Hygiene Commission, International Dental Federation

December 12, 1930

AN ATTEMPT TO PRODUCE VINCENT'S INFECTION IN THE WHITE RAT

S. J. RANDALL, D.D.S., *Major, Dental Corps, U. S. Army*

Research Laboratory of the College of Dentistry, Ohio State University, Columbus, Ohio, P. C. Kitchin, B.S., M.S., D.D.S., in charge

On January 7, 1930, a three-months old white rat, which had become rachitic as a result of feeding it Steenbock's rachitic diet, but which, for some time prior to the date mentioned above, had been placed again on a balanced ration, described below, was etherized and inoculated, deep into the subgingival tissues around the upper and lower incisor region, from an acute case of Vincent's infection. The diagnosis of the case just mentioned was based on clinical and microscopical findings. Forty-eight hours after the initial inoculation, the animal was again etherized and a thorough survey made for evidence of clinical manifestations of the disease, but none could be noted. Several smears were also taken at this time, slides prepared, stained (according to Appleton and Bryant; text—Laboratory Guide in Bacteriology, page 139) and examined, but no spirochaetes or fusiform bacilli could be identified, although numerous fields of streptococcus and staphlycoccus were readily recognizable. Two days later the same process was repeated and results obtained as before, that is, no evidence of Vincent's infection could be observed.

Eight days after the inoculation it was noted that the animal had a marked inflammation of the left eye. On the following day the eyelids were closed; and owing to dried pus sealing the eyelids, they were separated with difficulty. Thinking it might be possible that we had accidentally infected the eye with Vincent's organisms, smears were taken from this area, stained, and examined for spirochaetes and fusiform bacilli, but these slides were negative.

No further attempts were made to inoculate this white rat for a period of six weeks, when it was again inoculated as before from another known acute case of Vincent's stomatitis occurring in a patient who came to the College Dental Clinic for treatment. Smears were again taken at different intervals subsequent to this inoculation, but they were always found to be negative for Vincent's organisms.

This white rat was cancelled eighty-one days after starting this project, and a thorough survey and examination made in an effort to determine clinical evidence of Vincent's infection, but none could be observed. It was also decided to attempt to determine whether the addition of vitamin D (in the form of irradiated ergosterol—Squibb's Viosterol) to a rachitic diet would be a factor as regards susceptibility to Vincent's infection. This was started on February 18, 1930. Two adult and healthy white rats were isolated in cage No. 1 and placed on Steenbock's rachitic diet. At the same time, two other white rats from the same litter were isolated in cage No. 2 and placed on the rachitic diet mentioned above, plus 0.1 per cent of Viosterol. The two groups of rats were kept on these diets for one month, when they were all inoculated with Vincent's organisms taken from an acute case of the disease, which had been diagnosed as such both clinically and microscopically. Since these organisms were immediately transferred from the patient to the animal, it is believed that they were vital and potent when implanted into the subgingival tissues of the rat, as not over thirty seconds elapsed during the transfer from patient to animal.

After a period of three days the animals were etherized, a careful oral examination made, and two smears from each animal stained (using the same technique as that mentioned above) and examined for Vincent's organisms, but none could be found. This same procedure was repeated at the end of seven, eleven, and fourteen days, and in every case negative results were obtained. The two groups were cancelled five weeks after the inoculation, when another careful check was made and smears taken from suspected areas, but no spirochaetes or fusiform bacilli could be identified.

From results obtained in the foregoing experiments, it is believed that the white rat is not susceptible to Vincent's infection when fed on a balanced ration consisting of the following parts per hundred: Corn meal (yellow), 70.0; lard, 14.7; dried milk, 7.0; sodium chloride, 0.7; cod-liver oil, 0.6; meat scrap, 7.0. The results indicate that the albino rat is not susceptible to Vincent's infection when his physical condition and resistance are greatly lowered on a deficient diet such as Steenbock's rachitic diet. E. H. Wagner and her co-workers have also noted that species immunity to Vincent's infection is shown by kittens on a deficient diet. See the *Journal of Dental Research*, 1930, x, pages 591–597; October.

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Scientific Proceedings of the Chicago Section[1]
October 16, 1930

RUDOLF KRONFELD, M.D., *Editor*

Meeting held at the Medical and Dental Arts Club, 185 N. Wabash Avenue, Chicago, October 16, 1930. *Chairman:* Stanley D. Tylman, B.S., D.D.S., M.S., F.A.C.D., President of the Section.

Scientific program

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Abstracts

I. OCCUPATIONAL DERMATITIS COMPLICATED BY FOOD ALLERGY

B. Z. Rappaport, M.D., Medical School, University of Illinois, Chicago, Ill.

A dentist had exfoliation of the epidermis of the fingers of both hands for nearly four years. The condition was treated first as a fungus infection, and later as an occupational dermatosis, with only temporary improvement. Protein tests revealed hypersensitiveness to beans. Elimination of beans from the diet resulted in a gradual improvement until the lesions practically cleared completely.

II. FINDINGS IN RELATION TO RESORBING DECIDUOUS TEETH[2]

Lloyd B. Urban, B.S., Department of Histology, Northwestern University Dental School, Chicago, Ill.

Three things were shown which seem to have received little consideration in studies of the resorbing deciduous teeth; namely, pro-

[1] The last preceding series of abstracts of the proceedings of the Chicago Section was published in the *Journal of Dental Research:* 1930, x, p. 443.

[2] Presented by title only at the meeting of the Chicago Section on May 8, 1930: *Journal of Dental Research*, 1930, x, p. 445. Presented in full at this meeting. The complete paper will be published in the succeeding issue of the *Journal of Dental Research*.

liferation of the epithelial attachment along the root surface, recession of the gingivae, and lengthening of the clinical crown. Rather wide spread and severe damage, often accompanied by infection, occurs in the gingivae and in the enamel epithelium of the permanent tooth through prolonged retention of the deciduous fragment.

III. RADIOGRAPHIC AND HISTOLOGIC REPORT OF A CASE OF EXTENSIVE ROOT RESORPTION

Emil Mueller, M.S., D.D.S., Northwestern University Dental School, Chicago, Ill.

The patient lost a large number of her teeth within a period of less than a year. During this period x-ray pictures were taken at frequent intervals. In addition to careful and frequent clinical examination of the oral cavity, the patient was subjected to thorough physical examination by Dr. H. R. Rony, M.D., of the Northwestern University Medical School. Upon the basis of his findings a treatment was undertaken which, to date, appears to have checked the progress of this rather devastating process. Teeth were also extracted at various intervals and studied histologically. These together with the x-rays revealed very extensive and rapid resorption of the hard substances of almost all the teeth. After the general treatment had been started, these resorptions were to a great extent repaired by cementum and bone.

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Scientific Proceedings of the New York Section[1]
December 10, 1930

BISSELL B. PALMER, D.D.S., F.A.C.D., *Editor*

The tenth anniversary of the establishment, in New York, of the International Association for Dental Research, and of the New York Section, was celebrated at the Columbia University Club, 4 West 43rd Street, New York City, December 10, 1930. *Chairman:* Leuman M. Waugh, D.D.S., F.A.C.D., President of the Section.

Letter from Dr. J. Leon Williams, First President

Dr. J. Leon Williams, one of the founders of the International Association for Dental Research, and its first president, having been unable to attend the meeting, sent to President Waugh the following communication, which was deeply appreciated:

"Unfortunately, my health has been in such a precarious condition during the past two or three years that I have largely lost touch with the specific activities of our organization. But I do know in a general way what the organization has accomplished during the decade which closes tonight. It has probably contributed to the literature of our profession a larger number of papers which may deservedly be called scientific in character than any other dental organization in the country. But I would not esteem these meritorious contributions as the greatest work which the Society for Dental Research has accomplished. First and foremost I would place the stimulus its influence has exerted in the cultivation of the scientific spirit, *the scientific attitude of mind*, throughout the profession. A general scientific attitude of mind of a large body of men in any profession or calling is of enormously greater importance than any particular contribution to that profession, and

[1] The last preceding series of abstracts of proceedings of the New York Section was published in the *Journal of Dental Research:* 1930, x, p. 265.

for this reason, among others; pseudo-science, of which there is more than a little still flourishing, finds no soil in which it can grow among men who cultivate the scientific attitude of mind. On the other hand the merit of real scientific work meets with immediate recognition from those who have been trained to weigh and estimate the value of evidence.

"May I be permitted to refer to just one personal experience in illustration of this point. It is now more than thirty years since I said, in a paper read before a dental society in this city, as a brief summing up of a line of evidence presented: "If a tooth could be kept perfectly clean from the time of its eruption it would never decay." That statement seemed to me so completely obvious as to call for no further elaboration. But from that day to this the statement has been questioned by men more or less eminent in the dental profession. Truly, there is still great need for the foremost function of this society in the cultivation in the profession of a scientific attitude of mind.

"A large part of the credit for impressing the stamp of a scientific character on this organization is due to its founder, Dr. William J. Gies; and, as you all know, it is only one contribution out of many that he, who was not trained as a member of our profession, has made in the interests of higher and better dental education. I hope he finds some measure of reward in the fine results which have followed his work. I raise my glass in his honor. And to all of you, comrades in the search for truth, my kindest greetings and best wishes for the future."

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Abstracts

I. A BRIEF REVIEW OF THE HISTORY OF THE INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH[2]

William J. Gies, Ph.D., F.A.C.D., General Secretary, Columbia University

Ten years ago, today, our Association was founded to promote research in dentistry. The founders knew that research would be given cumulative encouragement and strength by a permanent union of men and women of similar aspirations for the advancement of dental science. The founders believed that meetings of the Association, besides giving aid and comfort to kindred spirits in inquiry, would facilitate coöperation in matters affecting research, and also provide occasion for recurrent expression of the kinds of constructive criticism upon which the most useful research flourishes. The founders realized that the meetings would also favor the best presentation of the results of research; would give to active investigators the benefit of analytic discussions of researches in progress; would encourage the younger workers to endeavor to merit the intellectual approbation of colleagues of more extended experience in research; would develop a morale and an ambition in dental research that otherwise might arise very slowly. The founders foresaw that the Association would stimulate achievement and productivity in dental research; and, by cultivating the scientific spirit, and by suitable publication of the proceedings, would improve public support of dental research, and would add further dignity, and impart finer quality and serviceability, to dentistry as a profession. These important expectations have been in process of gradual realization, and the establishment and efforts of the Association have been amply justified.

Our Association was founded at a meeting attended by twenty-one persons resident in New York City. Dr. J. Leon Williams presided;

[2] This paper is presented here in full.

Dr. Leuman M. Waugh was Secretary. "Articles of Agreement," effecting provisional organization, were adopted unanimously. Concurring in the Association's foundation—and although unavoidably absent from the meeting, participating in the establishment of the Association—were 4 persons resident in New York City, 14 in Boston, and 1 in Chicago, a total of 40 founders. During the past ten years the 40 founders have increased to a total membership of 228. The names of the founders follow:

Present at the meeting of organization

E. A. Bogue	R. G. Hutchinson, Jr.	Frederick A. Peeso
Adolph Berger	Thaddeus P. Hyatt	M. L. Rhein
Theodor Blum	Milo Hellman	James P. Ruyl
George W. Clapp	Victor H. Jackson	Paul R. Stillman
Henry S. Dunning	F. C. Kemple	Leuman M. Waugh
William J. Gies	Arthur H. Merritt	J. Leon Williams
Henry W. Gillett	Bissell B. Palmer, Jr.	J. Lowe Young

Unavoidably absent from the meeting of organization, but concurrent; formally elected to membership, as founders, by those present at the meeting

Resident in New York City.—Herman E. S. Chayes, William B. Dunning, M. I. Schamberg, Frank T. Van Woert.

Resident in Boston.—Robert R. Andrews, Lawrence W. Baker, George A. Bates, Fred R. Blumenthal, Percy R. Howe, A. Leroy Johnson, V. H. Kazanjian, L. M. S. Miner, William Rice, Alfred P. Rogers, H. Carlton Smith, Eugene H. Smith, Kurt H. Thoma, George H. Wright.

Resident in Chicago.—Frederick B. Noyes.

Of the 40 founders, 9—Andrews, Bates, Bogue, Jackson, Peeso, Rhein, Carlton Smith, Eugene Smith, and Van Woert—have preceded us into membership in the largest of all associations. I am confident their spirits are with us tonight.

The meeting at which the Association was founded had been preceded by correspondence that facilitated provisional establishment in accordance with a plan by which the Association would be promptly converted into a permanent international federation of autonomous local societies. On December 17, 1920, a meeting was held in Boston, where additional persons resident in Boston and Chicago were elected to membership. On December 30, 1920, a meeting was held in

Chicago at which additional persons resident in Chicago were elected to membership. On May 27, 1921, the groups in these three cities, by adopting the Constitution now in force, formally created the New York Section, the Boston Section, and the Chicago Section, as of the respective dates of the first meetings of the Association in these three cities. During the past ten years this total of 3 sections has become 15. Five different nations are now represented in the membership. The new sections were organized in the following sequence.

1921—Toronto
1923—Ann Arbor
1924—San Francisco
1928—Halifax, Minnesota, Philadelphia, Pittsburgh, St. Louis
1929—Vienna
1930—Cleveland, New Haven, Winnipeg

The membership of the sections ranges from 4 in Cleveland to 40 in New York. The local activity of the sections is indicated by the number of their meetings, which in 1929–30 was a total of 39—an average of nearly three per section. The number of the local meetings is steadily increasing, as interest and capacity in research expand in each geographical center.

The Constitution of the Association created and maintains an international body composed of national (general) divisions containing intranational (local) sections. The divisions and sections are autonomous in accordance with the limitations of ordinary federation. Members, who can be elected by the Association at annual meetings only, may organize divisions in any nation and sections in any division. Anyone who has conducted and published an account of a meritorious original investigation in dental science or art, or in any of the sciences contributory thereto, is eligible to membership. A Council, consisting of elected representatives of the national divisions, serves as an executive committee during the intervals between general meetings, which are devoted primarily to the presentation and discussion of papers on research by the members and their guests. Article III prohibits commercial relationships between the Association on one side and individuals or organizations on the other.

In addition to three preliminary meetings for purposes of organiza-

tion, eight general scientific meetings of the Association have been held on the following schedule:

1922	December 21	New York City	(I)
1924	March 7	Chicago	(II)
1925	March 20	Chicago	(III)
1926	March 26	Chicago	(IV)
1927	April 19	New York City	(V)
1928	March 25–28	Washington	(VI)
1929	March 23–24	Chicago	(VII)
1930	March 24–26	Toronto	(VIII)

The ninth general meeting will be held in Memphis, on March 21 and 22, 1931. The general meetings have grown from that in 1922—an evening meeting at which only two papers were read—to the program in 1930, at which fifty-four papers were presented at four sessions on three successive days.

Two statistical matters suggest the intellectual quality of the recent work of the Association. The participants in the program of the last general meeting held the following *academic and professional degrees:* Bachelor, 22; master, 11; doctor of dental surgery (or doctor of dental medicine), 40; doctor of medicine, 19; doctor of philosophy, 5. The researches represented on that program were reported from these *universities:* Columbia (7), Dalhousie (2), Harvard (1), Hawaii (3), Johns Hopkins (1), Loyola—Chicago (2), Michigan (2), New York (3), Northwestern (3), Pennsylvania (4), Pittsburgh (1), Toronto (2), Tufts (1), Vienna (8), Western Reserve (4), Yale (5). Papers were also presented from these independent *medical schools or hospitals:* Fifth Avenue Hospital, New York (1), Jefferson Medical College (1), Mayo Clinic (1), Peiping Union Medical College (1), Philadelphia General Hospital (1).

The Association has no financial liabilities, but the treasury contains a surplus of about $500, which amount has been saved from the apportionment of $1 per member per year for general expenses. The *Journal of Dental Research* has paid all expense of publication.

Four years ago, in historical comment on our Association, I wrote in part as follows:[3]

[3] Gies: *Journal of Dental Research*: 1926, vi, p. 111.

"The Association was organized to promote research, nothing else; and is wholly impersonal in character and avowedly non-political in purpose. Its development has been slow but spontaneous; and its activities, however restricted, have been natural not artificial. It had not been forced to walk before it was able to stand. Although the Association has not been much stronger than the slowly developing spirit of research in the dental profession, and has shown unmistakable symptoms of recurrent rickets, nevertheless the food which dental research is now receiving is better balanced and more nutritious than it has been. A further improvement of the present diet, which contains increasing proportions of the vitamins of financial sustenance, will cause more rapid growth of dental research, and will also assure the Association's attainment of health, vigor, activity, and fecundity. It is hoped that additional sections will be organized in this country and Canada, and abroad."

These quoted statements seem to be pertinent today. Our Association exemplifies an impersonal ideal. Through the coöperation of accredited investigators in the sciences upon which dentistry rests and which it uses, the Association promotes the discovery of truth and the dissemination of new knowledge to the end that, irrespective of beliefs, opinions, policies, or traditions, oral health-service in all its aspects may be given its greatest possible usefulness for the whole of humanity. With this history behind us, and with this ideal before us, we may proudly and hopefully look forward to the evolution of endless decades in the growth and increasing public value of our Association.

II. STUDIES, IN THE RAT, OF SUSCEPTIBILITY TO DENTAL CARIES. 1. BACTERIOLOGICAL AND NUTRITIONAL FACTORS[4]

Theodor Rosebury, D.D.S., and Maxwell Karshan, M.A., Ph.D., Departments of Bacteriology and Biological Chemistry, College of Physicians and Surgeons, Columbia University, New York City

Sixty young rats were studied in three groups, in which were tested, respectively, (a) a low-calcium, vitamin D-free diet, (b) a vitamin C-free diet, and (c) a diet high in fermentable carbohydrate (all

[4] This work was conducted with the aid of a grant from the Commonwealth Fund for research in dental caries. Published in full in this issue: *Journal of Dental Research*, 1931, xi, p. 121.

"synthetic"), in each case with and also without the addition of human oral lactobacilli, fed in an adhesive paste. The mouths of all rats were cultured at intervals for aciduric organisms.

The presence of rickets in the first group was ascertained by line tests and ash determinations on the tibias. Aciduric bacteria similar to *Lactobacillus acidophilus* were found to be a normal inhabitant of the rat mouth, their presence being constant, and independent of the feeding of human lactobacilli. No dental caries was produced, in spite of evidence of retention of food in molar pits, probably indicating the absence in these experiments of one or more factors in susceptibility to caries.

III. PATHOLOGICAL CHANGES IN THE TEETH OF RATS PRODUCED BY SYNTHETIC DIETS[5]

Theodor Rosebury, D.D.S., and Maxwell Karshan, M.A., Ph.D., Departments of Bacteriology and Biological Chemistry, College of Physicians and Surgeons, Columbia University, New York City

A "synthetic" diet low in calcium and free from vitamin D was found to produce, in the teeth of rats, changes that were ascribable to deficient calcification, as follows: The odontogenic zone is abnormally broad in the dentin of both molars and incisors, and large calcified globules appear in the body of the dentin. The enamel of the molars, and the fully formed enamel of the incisors, are apparently normal; but the youngest enamel of the incisors, and the apical ameloblasts, present evidence of deformity. An alteration in the staining quality of the alveolar bone occurs. The pulps, including the odontoblasts, remain normal.

Other "synthetic" diets, including a vitamin C-free diet, and diets in which vitamin C was supplied by 2 cc. of orange juice per rat per day, were found to give rise to changes in the pulps and dentin of the molars only, similar in some respects to the manifestations of latent scurvy in the teeth of guinea-pigs. These changes do not seem to be due to vitamin-C deficiency; their specific cause has not yet been determined.

[5] This work was conducted with the aid of a grant from the Commonwealth Fund for research in dental caries. Published in full in this issue: *Journal of Dental Research*, 1931, xi, p. 137.

IV. CONCERNING THE REACTION OF THE DENTINE TO EXTERNAL IRRITATIONS

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Sharply defined opaque zones in the dentine of ground sections of teeth have been regarded as being the result of such external irritations as decay, abrasion, or erosion. They are, however, not always in immediate connection with areas subjected to such irritations as, for instance, in roots. For this reason, it has been the author's belief that such changes were caused or accentuated by the technical procedure of grinding the specimen. New investigations have indicated that these opaque zones in the dentine are formed as a result of pathological changes in the pulp. This has been demonstrated by a specimen, a part of a superior maxillary, containing four teeth. The second bicuspid, and first and second molar, were partly carious and showed typical dark zones in both the crown and the root dentine. The unerupted third molar, on the other hand, containing a normal pulp, showed dentine of normal translucent appearance. The supposition, therefore, that the frequently noted opaque zones in the dentine are the result of technical manipulations, is apparently refuted by the condition observed in this specimen, as these four teeth were subjected to the same procedure at the same time.

It is therefore concluded that the opaque zones in the dentine probably indicate a derangement of the dental nutritional system as a result of pathological conditions of the pulp that may affect both crown and root areas.

V. TOOTH-BRUSH TRAUMA RECESSION: A CLINICAL STUDY

Isador Hirschfeld, D.D.S., School of Dentistry, Columbia University, New York City

The toothbrush, properly used, is without doubt the most potent practical means today of preventing various dental and periodontal diseases. On the other hand, a close observer will note that tremendous damage, dental or periodontal, may and frequently does accrue from improper manipulation of the brush. The incidence of such ill

results naturally increases in proportion to the spread of the toothbrushing habit. It is timely, therefore, that closer observations be made and recorded of the diagnosis, etiology, prevention, and correction (where possible) of such results. The essayist does not attempt in this paper to analyze the relative merits or shortcomings of the various standard methods of toothbrushing, but rather calls attention to damage produced by various irregularities in the carrying out of these methods as well as by anatomic abnormalities that require special consideration in the manipulation of the brush. He also stresses the importance of periodic inspection for any indications of damage resulting from improper brushing, as well as for a detailed checking up of the patient's handling of the brush. For the former purpose, a set of good record casts is invaluable for future comparison.

The essayist mentioned instances where different patients, soon after they had been properly instructed, fell into individual eccentricities in either the manner or sequence of brush manipulations, resulting rapidly in characteristic recession of the gingiva, toothbrush abrasion, cervical hypersensitiveness, or erosion. The tragedy of such end results is all the greater where the enthusiastic brushing is prompted by either vanity or special effort at prophylaxis. Photographic evidence showed progressive stages in recession due to toothbrush trauma, and also demonstrated that, with the corrected brushing, return of healthy gingival tissue toward its normal position and contour was made possible.

The following are some of the elements that induce gingival recession through toothbrush trauma:

(1) Anatomic abnormalities: (a) The dental arch—the more accentuated the convex contours, the greater the pressure per unit area at these points. (b) Malposition of individual teeth—as a tooth protrudes buccally to the arch, to such a degree do its investing tissues over the protruding part take on greater pressure per unit area. (c) Alveolar deficiency—over teeth so protruding, there is usually a corresponding alveolar deficiency; clinical observation seems to confirm the theory that, exposed to similar mechanical traumatization, gingiva covering a root surface directly will be effaced much more rapidly than that covering alveolar process.

2. Stiffness of bristles as influenced by locational sequence of brush-

ing—the stiffer the bristles, the greater the pressure they can exert; therefore, the most vulnerable parts of the arch; namely, accentuated contours, should be brushed last, i.e., when the bristles have become comparatively soft.

3. Size and briskness of the strokes—longer and more brisk strokes of the brush seem more destructive to the gingiva than the short and gentle strokes.

The essential points in the differential diagnosis of toothbrush trauma recession in its pure form are:

1. Its restriction generally to the labial or buccal side.

2. Its accentuation opposite individual teeth, to a degree according to their protrusion outside the dental arch.

3. The recession is most marked in the area first attacked by the brush, especially where the dental arch is normally formed. This may be corroborated by a toothbrushing demonstration by the uninformed patient.

VI. THE HISTO-PATHOLOGY OF THE PULPS OF TWO TEETH THAT HAD NO OCCLUSAL ANTAGONISTS

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The upper right central, lateral, and cuspid had no occlusal antagonists for over eight years and were therefore more or less functionless. These teeth never had been affected by dental caries, and the lesions of the pulp cannot be attributed to some previous infection or irritation. The pulps of the central and cuspid were studied because they were easier to handle. On cracking open the central, the pulp seemed abnormally large for the patient's age—about 45 years—and appeared to be hard and fibrous. The cuspid pulp was also large but more gelatinous and mucoid.

The pulps were fixed in Bouin's picro-formol fluid, dehydrated, cleared, embedded in paraffin, cut five microns in thickness, and stained with Masson's trichrome stain. The interesting features were the vital appearance of the odontoblasts, the infiltration of the original pulp-tissue with fat, the hypertrophic appearance of the nerves, and the degeneration of the nerve fibres.

VII. CALCIFICATION OF TEETH AND BONES ON RACHITIC AND NON-RACHITIC DIETS[6]

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In a previous report[7] it was shown that a severe rachitic diet which produced marked reduction in the ash, calcium, and phosphorus in the tibia bones of young rats, failed to cause significant changes in the incisor teeth of the same animals. The diet used, a modification of the Steenbock diet, consisted of yellow corn meal 66 per cent, wheat gluten 20 per cent, egg albumin 10 per cent, sodium chloride 1 per cent, calcium carbonate 3 per cent, spinach 10 grams per rat per day, and water *ad lib.* This diet is high in content of calcium and low in phosphorus. In the present experiments the same diet was used except that the $CaCO_3$ was removed, resulting in a diet low in both calcium and phosphorus. Also in another series KH_2PO_4 at 2 and 4 per cent levels was added, giving a diet low in calcium and high in phosphorus.

These diets gave results of a similar nature. The ash, calcium, and phosphorus of the incisor teeth were reduced approximately 10 per cent, whereas the tibia bones gave a reduction of about 30 per cent. When cod-liver oil was added (2 per cent) to the food of rats that had been on the deficient diets for about two months, the ash, calcium, and phosphorus of both teeth and bones were restored to normal.

VIII. MEDICO-DENTAL CASE RECORDS: IX. MYOCARDITIS[8]

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The patient, 62 years of age, had been troubled with shortness of breath for about a year, and gave a definite family history of cardiac disease—past medical history negative. Three months before her

[6] Part of this work was conducted with the aid of a grant from the Chemical Foundation for research in biochemistry.

[7] Karshan: *Journal of Dental Research*, 1930, x, p. 267.

[8] Published in full in the *Journal of Dental Research*: 1930, x, p. 675.

second entrance into the hospital, she was admitted principally for rest after a first acute attack of dyspnoea. The then existing cardiac irregularity and dyspnoea improved with rest, but the initial attack seemed to precipitate the continual subsequent disturbance. She had had digitalis continually. It seemed likely that she had an attack of coronary thrombosis as her initial condition, and the auricular fibrillation from which she suffered, were due to the involvement of the conducting mechanism in the infarct. She rallied from this attack, except for the fibrillation, which continued. In an attempt to remove any toxic influence upon the myocardium, she was admitted to the hospital for the second time for the removal of the infected teeth. At this time she showed no signs of *acute* illness but seemed to be in a fair state of health. The findings of the physical examination revealed no symptoms severe enough to contraindicate the operation for the removal of the infected teeth, nor was she in any sense considered a poor risk. Certainly patients with more advanced cardiac decompensation, and actually bad risks, have had more severe operations and have made uneventful recoveries; yet in this patient two slightly infected teeth were removed, and death resulted in a little more than thirty-six hours. Her final attack might have been due to another coronary thrombosis. Her symptoms of shock might have accompanied this condition. That such a severe exacerbation might result from toxic or infectious causes alone, considering the pathological condition of the teeth, seems unlikely.

The emotions of fear and anxiety had certainly disturbed the patient's equananimity, and it is not improbable that nervous influences were predisposing factors in the fatal termination. The following general conclusions were drawn:

1. The fatal exacerbation might have been caused by the direct effect of toxic or infectious absorption upon the parenchyma cells of the heart or the heart muscles, or upon the visceral nervous system. It seems more likely that the severe functional disturbance depended chiefly upon the nervous influences of the symptom-complex activated by the major emotions of fear and anxiety.

2. Functional disturbances of the heart may be exaggerated by nervous influences, of which the symptom-complex of fear-and-anxiety neurosis are important manifestations.

3. Sufficient study and *individual* consideration frequently is not given to the neurotic or psychotic patient.

4. The removal of chronic periapical foci of infection in patients with manifestations of secondary lesions is usually an operation of choice. Therefore, operation should proceed only after a careful study of the patient's general condition and then with extreme circumspection; and at the initial operation perhaps only one tooth should be removed.

STERILIZATION OF DENTAL INSTRUMENTS

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I. INTRODUCTION

A thorough knowledge of sterilization methods, their correct use, and their limitations, is of inestimable value to the dental practitioner. As the laity is becoming more familiar with the means of germ destruction, they are more apt to scrutinize the methods of sterilization employed by the dentist. As an asset, therefore, in developing his practice, it behooves the dentist to acquire the necessary information relative to these procedures. Furthermore, when one considers the important rôle that sterilization assumes in malpractice suits, the value of a fundamental knowledge of sterilization procedures is evident. The employment of sterile instruments, as a means of preventing infections, has been advocated so frequently that no further discussion of this matter is required at this time. Much is often taken for granted as to the efficacy of certain methods of rendering objects free from living microörganisms. This is not entirely the fault of the dentist, but is due rather to the inadequate and perhaps even erroneous instruction he has received. In the more general sources of information, one finds that little attention has been given to the factors that influence germicidal activity. In the following pages the writer attempts to give data on the more common means of sterilization, as obtained from his own experience and that of others.

II. CONDITIONS AFFECTING STERILIZATION

Procedures by which sterilization is accomplished may be divided into two main groups; namely, those in which destruction of microorganisms is effected through the use of chemicals, and those in which heat is used. The chemicals most commonly employed may be classified as (a) salts of the heavier metals, such as mercuric chloride; (b) members of the coal-tar group, such as phenol, lysol, and tricresol; (c) gaseous disinfectants, such as oxygen, formaldehyde, and chlorine; and (d) miscellaneous compounds like alcohol, chloramines, and—of more recent origin—metaphen and hexylresorcinol. Before discussing

TABLE 1

Data on germicidal power of some antiseptics

ANTISEPTIC	STAPHYLOCOCCI		STAPHYLOCOCCI	
	Suspended in water		Suspended in 50 per cent horse serum	
	Killed	Not killed	Killed	Not killed
Phenol	1:250	1:500	1:50	1:100
Salicylic acid	1:2500	1:5000	1:100	1:250
Hydrogen peroxide	1:3500	1:8000	1:1700	1:2500
Iodine	1:100,000	1:1,000,000	1:1000	1:2500
Mercuric chloride*	1:5,000,000	1:10,000,000	1:10,000	1:25,000
Silver nitrate*	1:1,000,000	1:10,000,000	1:10,000	1:25,000
Sodium hypochlorite	1:500,000	1:1,000,000	1:1500	1:2000
Chloramin-T	1:500,000	1:1,000,000	1:2000	1:3000

* No antidote used in experiments to prevent action of traces of salts in subcultures.

the germicides in detail, some of the factors upon which the success of disinfection depends will be considered.

Medium in which organisms are present. If microörganisms are present in a medium containing a large amount of organic matter, a germicide must generally be used in a more concentrated form and also for a longer period of time. According to Rosenau (15), Behring found that mercuric chloride in a dilution of 1:500,000 was sufficient to destroy the vegetative forms of the anthrax bacillus in a few minutes when the latter were suspended in water. However, when these organisms were in broth, a strength of 1:40,000 was necessary; when in blood serum, a dilution of 1:2,000 was not always

sufficient in the same period of time. Dakin and Dunham (5) give the data in *Table 1* concerning the action of various chemicals upon organisms in suspension in water and in 50 percent horse serum. They placed one drop of a 24-hour broth-culture of staphylococcus in a total volume of 5 cc. of sterile water or in 5 cc. of 50 percent horse serum. The antiseptics listed in *Table 1* were then added in sufficient quantity to give the indicated dilutions. After a period of two hours all mixtures were tested to determine whether any organisms survived. From the data in *Table 1* it is evident that the presence of organic matter greatly reduced the germicidal power of each antiseptic. Dakin found that when pus was present, the results were even less favorable.

Temperature. In a manner similar to all chemical reactions, heat influences germicidal action favorably. According to Rosenau (15), Heiden found that the spores of a strain of anthrax, which had not been destroyed in thirty-six days in 5 percent phenol, were killed in the same concentration in thirty minutes when the temperature was raised to 55°C. Chick (3) also found that the velocity of disinfection was increased with rise in temperature. In some instances with each increase of ten degrees in temperature, germicidal efficiency increased from two to eight times.

Dilution of disinfectant. An efficient germicide, when in a dilution of 1:1000, should not be added to another liquid and thus be further diluted. A more concentrated dilution should be used, so that after addition to the liquid to be disinfected, the concentration will be at least 1:1000.

Moisture in organisms. Organisms in a dry state are able to resist some germicides for a longer period of time than when in a moist condition. This is especially true with alcohol in its higher concentrations. As a means of overcoming this factor, the germicide must be allowed to act for a longer period of time.

Type of organism. Some disinfectants are more germicidal towards certain organisms than others. Phenol, for example, has practically no action upon spores, whereas it is very efficient in destroying vegetative forms. The spores of some strains of anthrax have been known to resist 5 percent phenol solution for so long as forty days.

Period of exposure to germicide. No germicide causes the death of

organisms instantaneously. The minimum period of exposure for this purpose is dependent upon the amount of associated organic matter, dilution of the germicide, type of organism, prevailing temperature, etc. A germicide, to be effective, must be able to penetrate to the bacterial protoplasm. For organisms surrounded by organic matter, oily films, or protective capsules, the time required for penetration may be much longer than for organisms suspended in water. It will therefore not suffice merely to dip instruments in a germicide to render them sterile, for the bacteria may be protected by saliva, pus, or blood, or may be in a dry condition.

III. COMMON GERMICIDES

Mercuric chloride in a dilution of 1:1000 is an excellent germicide, since it acts upon both vegetative and spore forms. However, in the presence of organic matter, its germicidal activity is markedly decreased. As shown by Dakin and Dunham (5), although a dilution of 1:5,000,000 destroys staphylococci in water within two hours, yet a dilution of 1:10,000 is necessary when the organisms are suspended in 50 percent horse serum. It combines with organic matter, especially protein material, producing insoluble compounds. Mercuric chloride, therefore, should not be used for the disinfection of blood, pus, or saliva. Because of its corrosive action it is unsatisfactory for instrument sterilization. Dobowsky (6) found that corrosion of instruments may occur in as short a time as twenty-five minutes, and that burs are attacked in a shorter time.

Phenol (*carbolic acid*) is an efficient germicide when limited to non-spore-formers. Rosenau (15) stated that a 1 percent or even 2 percent solution has no certain effect upon anthrax spores. A 3 percent solution requires seven days, a 4 percent solution three days, and a 5 percent solution two days, to kill anthrax spores. Investigators have reported that spores from certain strains of anthrax were still viable after immersion in 5 percent solution for as long as forty days. Phenol may be used for the disinfection of excreta and saliva, although it has some affinity for protein matter, thereby losing some of its germicidal activity. It is generally used in 2 to 5 percent aqueous solutions, in which concentrations it destroys non-spore-formers within five minutes when they are readily accessible. It should not be used for

the sterilization of instruments since it tends to corrode them within a short time. Corrosion may be prevented to some extent by placing the instruments in 95 percent alcohol for several minutes after their removal from the phenol solution, and then wiping them dry.

Tricresol. The cresols are found in coal tar along with phenol, and are very efficient germicides. Tricresol consists of a combination of ortho-, meta-, and para-cresols. In germicidal activity it is at least three times as powerful per volume as phenol. It has further advantages over phenol in that its activity is not diminished by the presence of organic matter. Tricresol also destroys spores. It is generally used in 2 to 3 percent solution. Staphylococci and streptococci are destroyed within five minutes. According to Dobowsky (6) tricresol does not corrode instruments when in contact for periods as long as forty-eight hours.

Lysol is a liquid containing about 50 percent of cresols in addition to a neutral potassium soap. It acts like tricresol, being three times as germicidal as phenol. Lysol also acts well in the presence of organic matter, and destroys spores. It is used in dilutions of 1 to 3 percent, and does not corrode instruments within forty-eight hours. One disadvantage of both tricresol and lysol is their unpleasant odor.

Formaldehyde, and watery solutions of it prepared from formalin, have been used extensively for sterilization purposes. It has been advocated for the sterilization of the handpiece. I recently tested an apparatus consisting of a cylinder at the bottom of which cotton saturated with formalin had been placed. Sterilization was thought to occur by the action of formaldehyde vapor, from the formalin, upon the handpiece inserted into the cylinder. The time in which sterilization was said to occur was ten minutes. The efficacy of the apparatus was tested in the following manner. Small loopfuls of a saline suspension of both staphylococci and B. coli were allowed to dry upon the inner surfaces of sterile pieces of glass tubing, which were then placed in the container and individual pieces removed in 10, 20, 30, and 60 minutes, respectively. A loopful of sterile water was then placed upon the dried organisms, which were then emulsified and transferred to extract broth. Growth was obtained even after an exposure for one hour. All growths were examined morphologically and culturally to prove that no contamination had occurred. Has-

seltine (10), in tests upon the destructive action of formaldehyde, obtained similar results. Rosenau (15) has written: "The secret of successful disinfection with this substance is to obtain a large volume of gas in a short time. . . . . Formaldehyde gas under ordinary circumstances possesses small powers of penetration, it cannot be depended upon to accomplish more than a surface disinfection. . . . From its watery solution at ordinary temperatures formaldehyde gas is given off very slowly and in very uncertain quantity." These statements explain fully the cause of sterilization failure with the above apparatus. According to Dobowsky (6), "disinfection through gas formation of formaldehyde from tablets has no particular significance in the disinfection of instruments. Owing to its unreliability and the difficulty of control, it should be rejected."

Watery solutions of formaldehyde are also used to destroy microorganisms. These solutions are prepared from formalin, which is approximately a 40 percent solution of formaldehyde in water. The formalin is generally diluted with water to make from 3 to 5 percent solutions. Rosenau (15) stated that 4 percent solution of formalin in water is equivalent to about a 1:1000 solution of mercuric chloride, and is superior to 5 percent phenol solution. Hailer (6) found that 3 percent solution did not destroy staphylococci in thirty minutes, but required at least sixty minutes. When the staphylococci were dried upon shells, it required forty minutes for their destruction with a 4 percent solution. Dobowsky (6) maintained that if watery formaldehyde solutions are used, it is necessary, in order not to prolong the time necessary for disinfection, to use higher concentrations (more than 5 percent). Although formalin attacks various metals containing iron or steel, it does not corrode copper, brass, or nickel. If the instruments are well nickeled, they may be placed in formalin. An addition of soap, generally a potassium soap—2 to 4 percent—eliminates the danger of rusting and, according to Hailer (6), also multiplies the bactericidal effect about 3.5 times.

Alcohol. The assumption that alcohol is an efficient germicide is not supported by scientific observation. The mere dipping of instruments into alcohol, or wiping them with cotton saturated with this fluid, should be frowned upon in view of the following facts. Ninety-five percent alcohol is less germicidal than alcohol of lower concentra-

tions. According to Christian (4), "the maximum killing-power of alcohols is in solutions of 40 to 50 percent and beyond these limits its action declines." Harrington and Walker (9) have done extensive experiments to determine the efficiency of alcohol as a germicidal agent. Their method was to immerse sterile threads in 24-hour broth-cultures of non-spore-forming organisms such as staphylococcus, B. coli, B. typhosus, B. pyocyaneus, and B. diphtheriae. These organisms were used in both moist and dry condition. Harrington and Walker found as follows:

1. "Against dry bacteria, absolute and ordinary commercial alcohol (95 percent) are wholly devoid of bactericidal power, even with twenty-four hours contact; and other preparations of alcohol containing more than 70 percent, by volume, are weak in this regard, according to their content of alcohol; the stronger in alcohol, the weaker in action.

2. "Against the commoner, non-sporing, pathogenic bacteria in a moist condition, any strength of alcohol above 40 percent, by volume, is effective in five minutes, and certain preparations within one minute.

3. "Alcohol of less than 40 percent strength is too slow in action or too uncertain in results against pathogenic bacteria, whether moist or dry.

4. "The most effective dilutions of alcohol against the strongly resisting (non-sporing) bacteria, such as the pus organisms, in the dry state, are those containing from 60 percent to 70 per cent, by volume, which strengths are equally efficient against the same organisms in the moist condition.

5. "Unless the bacterial envelope contains a certain amount of moisture it is impervious to strong alcohol; but dried bacteria, when brought into contact with dilute alcohol containing from 30 percent to 60 percent of water by volume, will absorb the necessary amount of water therefrom very quickly, and then the alcohol itself can reach the cell protoplasm and destroy it.

6. "The stronger preparations of alcohol possess no advantage over the 60 percent to 70 percent preparations, even when the bacteria are moist; therefore, and since they are inert against dry bacteria, they should not be employed at all as a means of securing an aseptic condition of the skin."

Nye and Mallory (13), in an investigation to determine the means of infection in two patients who died from gas gangrene subsequent to operation in a Boston Hospital, found that the Bard Parker knife used in these operations had formerly been employed in removing

macerated tissue from a patient having a gun-shot wound. They found that the method of sterilizing these knives was to wash them in hot soapsuds and rinse in scalding water. Before use the knives were placed in 70 percent alcohol for a period of five to twenty-five minutes. As a result of their investigation it was shown that the organism (B. aerogenes capsulatus) was not destroyed by immersion for one hour in 70 percent alcohol.

From the above observations, we may conclude that alcohol should not be used in a concentration greater than 70 percent by volume, and that it should not be used to destroy spore-forming organisms.

Alcohol and glycerin. A mixture of two parts of alcohol and one of glycerin is often employed to sterilize hypodermic needles and syringes. This mixture is not more germicidal than 70 per cent alcohol in water, but probably less. Rosenau (16) found that glycerin has practically no antiseptic value. He was able to show that glycerin in a 50 percent dilution possesses more germicidal activity than in any other dilution. He found that pus cocci and B. coli were able to survive in it for at least two weeks. Spore-forming organisms were still viable even after two hundred days. According to Dobowsky (6), the addition of glycerin, while preventing rusting, also decreases the bactericidal power of the germicide. In some experiments I have performed with this mixture, I found that when Staphylococcus aureus and B. coli were suspended in water, and then mixed with the above solution, they were destroyed within five minutes. However, the organisms in a dry condition were not destroyed by immersion in alcohol-glycerin for thirty minutes.

Alcohol and the flame. Sanderson (17) performed some experiments to determine the sterilization effect of placing instruments in alcohol and then burning off the latter. She found that non-spore formers were destroyed, but that the spore formers, such as B. tetani, B. botulinus, B. sporogenes, and B. anthracis were not killed. When a mixture of ethyl alcohol and formalin (3:1) was used, the results were more satisfactory since growth was obtained in only two out of thirty-five tests on sporing organisms. However, when grease, oil, or blood was present, the results were not favorable. Sanderson further determined the temperature attained in the ignition of the fluids. The bulb of a thermometer was placed in these solutions, then removed, and the alcohol adherent to the thermometer ignited. Ethyl alcohol

gave a temperature of 60°C., and the formalin-alcohol mixture a temperature of 133°C. Sanderson stated that the flame in the latter test might not have been more intense, but burned for a longer time. She advised that when absolute sterility is required, the "burning off" of the instruments with an inflammable fluid cannot always be relied upon.

Metaphen. Metaphen is an organic compound containing 60 percent of mercury. According to Raiziss and Severac (14), the germicidal activity of metaphen is very much greater than mercuric chloride, mercurochrome, or phenol (about 500 times as strong as phenol). Metaphen, unlike mercuric chloride, precipitates albuminous material only slightly; and, since its mercury is in organic combination, metals, with the exception of aluminum, are not attacked or corroded. However, it must be remembered that aqueous solutions of metaphen, owing to the presence of water, eventually cause corrosion. In order therefore to eliminate this disadvantage, the manufacturers have placed upon the market a product called Metaphen-C to be used for instrument sterilization. This solution is so well buffered with alcohol and ethylene glycol that no corrosion and rusting was caused by continuous immersion for fifteen months. I have found that burs kept for seven consecutive days in Metaphen-C showed no tarnishing or rusting. No growth was obtained in subcultures prepared from dry staphylococci following immersion in Metaphen-C solution for a period of ten minutes. Before culturing, the Metaphen-C was removed and the organisms washed in sterile water. Controls treated in a similar manner, in the absence of a germicide, gave growth. No shorter periods of exposure were tested.

Hexylresorcinol ("S.T. 37"). According to Leonard and Feirer (12), "S.T. 37" consists of 30 percent glycerin and 70 percent water in which is dissolved 1 milligram of crystalline hexylresorcinol per cc. This solution possesses a surface tension of 37 dynes per cc., and represents the optimum composition of the solution investigated, since it is stable, non-toxic, and highly bactericidal. These investigators maintain that all the major types of pathogenic microörganisms are destroyed in less than fifteen seconds when in contact with this solution. Concerning its action upon instruments the manufacturers (18) state that "iron and steel instruments, if not protected by suitable plating

such as nickel and chromium, would be attacked slowly by water or aqueous solutions, including 'S.T. 37.' However, immersing in 'S.T. 37' for sufficient length of time to insure sterilization will not cause corrosion. It is understood that implements would not be stored while still wet."

I have found that burs kept for several days in this solution were not corroded. When however "S.T. 37" was diluted, a slight amount of rusting occurred within forty-eight to seventy-two hours. Staphylococci, in the dry state, were destroyed when exposed to "S.T. 37" solution for ten minutes. No shorter periods of exposure were tested.

IV. AGENCIES FOR HEAT STERILIZATION

The agencies for heat sterilization that are valuable to the dentist are boiling water, the hot-oil bath, and the high-pressure steam sterilizer.

Boiling water is without doubt the most practical and efficient means of sterilization for the average practitioner. Boiling for three minutes will destroy non-spore-formers such as staphylococci and streptococci; in some instances as short a time as one minute suffices to kill certain of these organisms. The spore-forming bacteria resist for a longer period the destructive action of moist heat. Although some spores survive the boiling for thirty minutes, they need not cause any alarm since they are probably all saprophytic in nature. According to Ford (7), anthrax spores are generally destroyed in ten minutes, while some strains are killed in four minutes.

A small amount of some alkaline substance such as sodium carbonate (1–2 percent) should be placed in the sterilizer to prevent rusting of the instruments. The addition of such substances also raises the boiling point several degrees. Heller (11) prefers sodium hydroxide (0.25 percent) because its use prevents the formation of a white scum upon instruments and eliminates the possibility of their becoming discolored. The desired strength of alkali can be approximated by dissolving a piece of the pearly-stick sodium hydroxide three quarters of an inch long per quart of water used. Heller found that when instruments were left overnight in this solution they did not tarnish.

Hasseltine (10), in an excellent paper on the sterilization of dental instruments, suggested the following procedures:

(1) All instruments and appliances should be rendered mechanically clean by washing in water with a brush or sponge.

(2) The following instruments and appliances should be kept in the slightly alkaline solution for ten minutes, at a temperature of 80° C. to boiling:

Artificial teeth used in measuring and matching
Burs
Chip blowers
Chisels
Excavators
Explorers
Files
Forceps (extracting)
Forceps (foil)
Hand pieces (for the engine)
Impression trays
Knives and lancets
Mallets (hand and automatic)
Mirrors (at 80° C., *but not in the boiling fluid*)
Mixing slabs
Mouth gags
Mouth piece (for saliva ejector)
Pliers
Pluggers
Polishing points and brushes (if not discarded after being used once)
Pyorrhea instruments
Reamers
Root elevators
Rubber-dam clamp and forceps
Saws
Scalers
Scratch wheel (in head of engine)
Spatulas, metal
Syringes, hypodermic
Syringes, water
Tongue-holding forceps

According to Hasseltine, when the mechanical construction of a particular instrument makes it difficult to remove the liquid after such treatment, the instrument should then be placed in 95 percent alcohol for ten minutes, dried, and oiled. In this manner rusting of the hand piece, for example, can be prevented. To insure complete drying, I suggest that the instrument be immersed in ether after removal from the alcohol.

Hasseltine also advises that instruments that would be injured by the temperature of boiling water, e.g., such as are made of tortoise shell or possess wooden handles, mirrors, and mounted stones, be placed for one hour in 5 percent phenol solution, then immersed in 95 percent alcohol to neutralize the phenol and to aid in drying. I have found that the better grade of mirror withstands repeated treatment

with boiling water over a period of several months before it must be discarded owing to discoloration.

Oil bath. The main disadvantages of sterilizing instruments in boiling liquid is the effect on the cutting edges, which become duller, and a certain amount of rusting occurs in time. Through the use of the oil bath, both dulling and rusting may be prevented. This method consists of placing the instruments and appliances in such oils as albolene or mineral oil, which are heated sufficiently to destroy spores. Appleton (1) stated that the hand piece should be first freed from moisture, especially the parts that come in contact with the bur. The bur lock should be left open before the hand piece is immersed in the mineral oil. He found that the spores of B. subtilis and B. anthracis were destroyed when the mineral oil was heated to 185° C. for five minutes; to 175° C. for ten minutes; or to 165° C. for fifteen minutes.

High-pressure steam sterilizer. Since this type of sterilizer is becoming more popular among dentists, a few words concerning it may not be amiss. No one engaged in the field of oral surgery can very well do without a high-pressure sterilizer for the sterilization of such articles as gowns, sponges, dressings, towels, and rubber gloves. In purchasing a pressure sterilizer, the location for the entrance of the steam to the chamber, and the outlet for the air, should be noted. Steam entering at the bottom of the sterilizing chamber tends to rise, and in so doing mixes with all of the air in the chamber, forming a steam-air mixture, and requiring an extra amount of steam over a prolonged period to insure complete removal of the air from the chamber. On the other hand, when it enters at the top, steam being lighter than the air, the latter is "blanketed" and forced out through the escape at the bottom of the chamber. The air should be expelled to prevent the formation of air pockets, which are poor conductors of heat, and also prevent the steam from gaining access to the material to be sterilized. The registrations of the thermometer, and of the pressure gauge located on the outside of the sterilizer, do not record the presence of air within the chamber.

That the thermometer and pressure gauge do not record the correct temperature of the articles undergoing sterilization has been shown by various investigators. Hallilay (8) found that drums packed with dressings and towels may have a temperature within the drum of only

97° C. while the thermometer records a temperature of 121° C. for the sterilizing chamber. The sterilizer should not be overpacked with material to be sterilized. Benton and Leighton (2) have shown that these objects should be so placed that the steam has ready access to them. These investigators packed flat bottles of water close together in the center of a sterilizer, and surrounded them with flasks containing water. The temperature of the bottles at the center was 105° C. while that of the flasks at the edge was 115° C. Therefore, in order to determine more accurately the correct conditions within the sterilizer, it is customary to use controls for this purpose. These controls may be either bacteriological or chemical. The sterilizer is controlled bacteriologically by the use of suspensions of spores which, subsequent to the "run," are cultured to determine their viability. The chemical controls may consist of indicators that change color at a specific temperature. An example of this type of indicator is the Diack tube, which at 121° C. changes from yellowish brown to red. Fusible alloys containing bismuth are also used. These alloys fuse at a specific temperature and may be made in various proportions so that fusion will occur at any temperature desired. It must be remembered, however, that the chemical controls are not as efficient as the bacteriological, since the former react to either moist or dry heat.

V. SUMMARY

Some of the more common methods of sterilization with chemicals and with heat have been discussed. The factors upon which successful sterilization are dependent have been emphasized. The conclusions that may be drawn from the data presented are these:

1. Successful use of various germicides is dependent upon such factors as (a) presence of organic matter, (b) period of exposure, (c) temperature, (d) dilution of germicide, (e) condition of organisms (moist or dry), and (f) spore formation.

2. Mercuric chloride is unsatisfactory for instrument sterilization owing to its corrosive action and poor germicidal activity in the presence of organic matter, such as blood, pus, and saliva.

3. Lysol and tricresol are three times as germicidal as phenol, and unlike phenol are promptly destructive of spores. Their aqueous solutions do not corrode instruments, even after immersion for several days.

4. Formaldehyde gas has not been practical for instrument sterilization since, at ordinary temperatures, insufficient gas is obtained from the watery solutions. The vapors are given off too slowly and in quantities too variable to be germicidal.

5. Formalin solutions should be used in concentrated dilutions (over 5 percent). It is advisable to add potassium soap (2 to 4 percent) to prevent rusting and also to increase germicidal activity.

6. Alcohol should not be used in concentrations higher than 60 to 70 percent. Dry organisms are not attacked as readily as moist organisms. Alcohol is not dependable for the destruction of spores.

7. Alcohol-glycerin mixture is no more germicidal than 70 percent alcohol. Possibly the addition of glycerin reduces its germicidal activity. Dry staphylococci were not destroyed by immersion for thirty minutes in this mixture.

8. Alcohol and the flame cannot be depended upon for the destruction of other than non-spore-formers. In the presence of grease, oil, and blood, the destructive action is questionable.

9. Metaphen-C is satisfactory, since no corrosion occurs and it possesses high germicidal power.

10. Hexylresorcinol ("S.T. 37") possesses high germicidal power. Corrosion of instruments occurs in aqueous solutions unless the instruments are promptly dried.

11. Boiling water containing alkali, such as sodium hydroxide, is the most satisfactory method of assuring successful sterilization. The disadvantages of this method are rusting and dulling of instruments. Rusting may be prevented by carefully drying the instruments daily.

12. An oil bath is an ideal agency for the sterilization of instruments, in which rusting and dulling of instruments are prevented.

13. A steam sterilizer, to be run efficiently, must not be overpacked and must not contain entrapped air.

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NEWER CONCEPTS OF FOCAL INFECTION[1]

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I. INTRODUCTION

The topic of *focal infection* concerns the dentist much more than the physician. For the physician it is, or should be, but one of the influences which affect health, whereas for the dentist, because of the limitations of his work and the ease with which teeth are injured by

[1] Given before the First District Dental Society of the State of New York, New York Academy of Medicine, November 3, 1930. Also given before the Los Angeles County Dental Society, April 15, 1930; Pasadena Dental Society, October 14, 1930; Metropolitan District Dental Society, Boston, October 29, 1930.

disease and therapeutic measures, it becomes at once the perspective by which the theory and practice of dentistry are now being judged. Thus one may advantageously paraphrase Byron:

> "Focal infection is of physician's work a thing apart.
> 'Tis dentist's whole existence."

Whether the future proves or disproves the truth of the principle of focal infection, the effect it has had upon dental thought and development will always remain. The great question concerning focal infection is: "What effect does it have on the body health?" There are three sets of opinions regarding this question and three answers: first, those which regard it as a tremendous influence in the production of disease; second, those which deny that it has any effect on the body as a whole; and last, those which give it some importance, the degree of which has not yet been established. European clinicians regard the principle of focal infection with skepticism. The greatest number of conservative and enthusiastic champions of it are found in our own country. It is essentially an American contribution.

In this article I wish to discuss some of the more important data concerning focal infection in the light of newer developments in physiology and the allied sciences. During the course of this exposition, it will be necessary to review critically some of the so-called definite and well-established data.

II. CLINICAL DATA AND LABORATORY DATA CONTRASTED

Focal-infection data may be considered under two sub-divisions: (1) clinical and (2) laboratory. *Clinical data* consist of case reports showing results obtained from the removal of foci of infection, while *laboratory data* consist of deductions made from experimental studies. These case reports almost universally agree that the removal of foci of infection produces distinct benefits in the treatment of disease. It is argued, therefore, that, since the removal of foci of infection aids or cures certain diseases, the foci must cause these diseases. Laboratory data disagree with this conclusion—in fact, the more carefully executed and controlled the experiments, and the more experienced the workers, the greater the discrepancy between the facts produced by these two types of study. It is contended that bedside material is very poor material for research purposes, because it is difficult to

control environmental influences and impossible to carry out duplicate series of experiments under identical conditions. Therefore, it is thought that laboratory evidence is more dependable. However, if clinical deductions are supported by long continued and careful observations they are just as reliable as those of the laboratory. Much of the clinical data is beyond reproach on this score. Why, then, does this wide difference of opinion as between the bedside and laboratory studies exist? There are many reasons, the most important of which are (1) the investigators themselves and (2) inherent sources of error in technique, which are either unrecognized or ignored.

1. Investigators

A well trained clinician is just as capable of carrying on a research problem as a full-time student, provided he or she confines the investigations to clinical problems. But when such a person attempts to study in another field such as bacteriology or physiology, which though related to clinical medicine is different in its concept and background, without first having had sufficient training in it, the liability to error begins. In the work on focal infection this objection is particularly noticeable. One finds dentists investigating and publishing data which, in order to be properly appreciated, require a thorough training in medicine; physicians studying a dental phase without dental training, or independently of guidance by a dentist; the pure scientists, such as bacteriologists or biochemists, drawing clinical inferences from their studies without the necessary bedside experience to judge the work properly. Although this subject should be studied from all possible angles, very few are the instances in which the combined efforts of all these investigators have been employed in any one problem in focal infection. Therefore, many of the so-called general facts, in reality, are only single phases of the whole subject.

2. Inherent sources of error in technique

This factor must be analyzed from two aspects: clinical and research. Clinically, one of the most prolific sources of confusion lies in the use of the terms *focus of infection* and *focal infection* as synonyms. In the term *focal infection* there are two separate and distinct ideas; namely, infection and focus of infection. *Infection* means the successful inva-

sion of the body or any of its parts by bacteria. A *focus of infection* is a circumscribed mass of tissue that harbors an infection. A *focal infection* is a systemic infection that has developed from a focus and continues to receive contributions from it. No one would be so bold as to deny that an appreciable length of time elapses between the establishment of a focus and the development of a focal infection. What, then, is to prevent the local defensive powers in the tissue that constitute the focus from blocking the dissemination of these products during this interval? Holman (21), calls attention to this fact, so often disregarded in practice, that a focus of infection may be latent or remain permanently inactive. It is clear that "focus of infection" and "focal infection" are entirely different in their true meanings.

Even granting that every focus produces a focal infection, how does one know that a particular focus is the one responsible for the ailment? This it is practically impossible to determine, since foci of infection may be present not only in the teeth, tonsils, and sinuses, but also in the middle ear, lymphatic glands, lungs, bronchi, gall bladder, colon, appendix, uterus, tubes, ovaries, prostate, seminal vesicles, urethra, rectum, anus, the nails, skin, and mucous membranes. I daresay that in a searching examination very few persons, if any, regardless of the state of health, would be found free from foci of infection. Therefore, when the statement is made that all the foci have been investigated, it probably does not include more than those which are easily accessible.

Clinicians have vested the laboratory with an atmosphere of infallibility, which it does not claim or justly deserve. In spite of the so-called scientific technique employed, errors cannot be prevented. It is only recently that research methods have been applied to problems in dentistry and medicine. The materials are either new, or old ones put to new uses, and there has not been sufficient time to learn all their peculiarities and reactions. Many laboratory facts, when applied at the bedside, prove disappointing, because the analogy between the human and laboratory sources of a similar reaction does not always hold. In the study of living beings, such as bacteria and animals, the fact that they possess hereditary tendencies and susceptibilities is often disregarded. My associate frequently states that everything lives in a world of its own, which is thoroughly individual and probably

as complex as ours. Therefore, one must take care not to confuse environmental reactions with those due to the conditions of the experiment.

An investigation of focal infection presents just such a problem. First, it deals principally with the streptococcus, an organism of wide distribution in nature and in the animal body, having close cultural and morphological similarity to other microörganisms, which may make its identification difficult. Second, it is only recently that investigators have realized the importance of considering the susceptibilities of the animals themselves, such as, for instance, the ease with which cardiac disease can be produced in young rabbits, the occurrence of peptic ulcers in animals used in other experiments, etc. The third consideration is the enormous complexity of man with his highly specialized tissues, reflexes, and defense mechanisms, to say nothing of the physiological influences of that little understood source, the mind. Therefore, it can be seen that one may not form conclusions from laboratory or bedside evidence alone.

III. FOCAL-INFECTION DATA CLASSIFIED

Focal-infection data can be best studied by classifying them into the following three groups: (1) bacteriological, (2) comparative importance of various foci, and (3) clinical.

1. Bacteriological data

The bacteriology of focal infection is mostly that of streptococci, although staphylococci, pneumococci, and influenza and colon bacilli are sometimes found. The intimate relation between focal infection and bacteriology offers a possible means of explaining how a focus of infection produces a focal infection. Up to the present time there have been four widely discussed theories:

(1) E. C. Rosenow's "elective localization."

(2) Karl F. Meyer's "opportunity, or organ selective action."

(3) "Anaphylaxis or protein sensitization."

(4) "Locus minoris resistentiae" or "point of lowered resistance."

In 1919 E. C. Rosenow (46) summarized his studies up to that time on the behavior of streptococci cultured from foci of infection. He made cultures from gingival, tonsillar, and dental foci, injected them into animals, and found that, in a considerable number, the same tis-

sues or organs were affected in the animals as those in the patients from whom the inoculations were taken. He called this phenomenon *elective localization*, and believed it was imparted to an otherwise non-specific organism by forcing it to grow in an atmosphere of diminished oxygen tension. Karl F. Meyer (33) disputed this conclusion on the ground that he had observed that streptococci were very prone to infect areas which had been injured either by disease, or by experimental or accidental trauma. He believed that the streptococcus is a *bacterium of opportunity*, and that it was the organ or tissue that decided the selection, rather than a specific property of the germ. These two theories do not explain the frequent observation that, in so-called focal infection, many inoculations made from the diseased tissues were sterile. Recently Cecil (7), collaborating with Nichols and Stainsby, has shown that a number of these fruitless results were due to improper methods. He succeeded in obtaining growths in some instances by changing the technique.

Where no growths are obtained, it is assumed that the bacteria in the focus elaborate a foreign protein which sensitizes a tissue, producing inflammation without the presence of microörganisms. This is called *anaphylaxis or protein sensitization*, and has been studied very extensively with disappointing results. McKenzie and Hangar (30) failed to find any relationship between skin reactions obtained with filtrates and extracts of hemolytic and non-hemolytic streptococci and any disease process. Derick and Andrews (17) obtained a positive skin reaction in 50 percent of normal rabbits tested with living and dead sedimented cultures of non-hemolytic streptococci. Routine skin tests of 119 patients in my office with bacterial antigens during 1923–1927 recorded only two that showed the typical reaction. Therefore, the anaphylaxis theory has not found any consistent experimental support.

The theory on the "*point of lowered resistance*" assumes that a focus of infection is not a disease-producing agency, but acts as a defective point in the defensive armor of the body through which the causes of the disease enter the system. Since this is such a broad statement, it offers no special items for agreement or denial.

Of these four theories, Rosenow's has received the greatest attention, with resulting strong support and bitter opposition. The strongest support of the hypothesis of elective localization is to be found in the

large number of cases of diseases which, as reported, have been improved or cured by the removal of foci of infection. · Numerous objections to his work, such as the use of large doses of cultures, direct injection into the blood stream, use of microörganisms at the height of their virulence, and inability of other investigators to agree on the interpretations of the findings, have been principally of a bacteriological character. In the tables Rosenow (46) submitted, several important clinical objections are to be found. It will be observed that the number of animals injected varied from 62 in the investigation of myositis and chronic arthritis, to 1 in appendicitis. He used 156 animals for nine disease entities making an average of 17 animals for each disease, a number which is quite small considering the vast territory covered by the experiment. It will be noted that the incidence of infection in the same part of the animal as that in the patient was 100 percent only in appendicitis, where but one animal was used. In the other experiments, the incidences of infection in the organs of the animals, other than those involved in the patients, were quite high; for instance, in the keratitis-iritis experiment, there was a 60 percent incidence not only in the iris and cornea of the five animals injected but also in the muscles, teeth and nerves, plus a 40 percent incidence in the joints. The chart used in tabulating the incidences was divided into 29 columns, one for each organ. The experiment on herpes zoster was carried out in 16 animals, which showed a localization in 20 of the 29 organs. Certainly this cannot be called elective localization; it is a septicemia. In the same experiment the localization for nerves was only 39 percent.

Another interesting fact in Rosenow's paper (46) was the high percentage of involvement of the muscle and joint systems in all the animals used. In the actual muscle and joint experiment, he used 62 animals, of which 73 percent showed involvement of these organs. In the peptic ulcer study with 24 animals, 16 percent showed muscle and joint lesions; in appendicitis with 1 animal, 0 percent involvement; neuritis, 12 animals, 58 percent involvement; herpes zoster, 16 animals, 56 percent; multiple sclerosis, 12 animals, 42 percent; transverse myelitis of the spinal cord, 15 animals, 7 percent; goiter, 9 animals, 55 percent; keratitis-iritis, 5 animals, 100 percent muscle and joint involvement. These figures weaken the stand that joint and

muscle inflammations are the result of elective localization. It might be suggested that such a high incidence in these organs is due to the fact that they contain terminal capillary systems, which are easily deranged by any traumatic agency, such as heat, cold, and over-use, as well as bacteria, and are, therefore, an easy prey to any powerful disturbance such as experimental inoculation. It is of interest to the dentist to point out that, in the myositis-arthritis, herpes zoster, and keratitis-iritis experiments, the teeth of the animals were also the sites of localizations to the extent of 11 percent, 25 percent, and 60 percent, respectively, answering in the affirmative Dr. Ottolengui's query: "Does an infection of any other part of the body ever produce an infection in the teeth?" Thus it can be seen that bacteriology offers very little positive information regarding the mechanism of focal infection, even though it thoroughly establishes the existence of the focus.

2. Comparative importance of various foci

Certain foci of infection, because of their surface exposure, blood supply, structure and functional activity, are more likely to produce systemic manifestations than others. These factors, especially surface exposure, are rarely considered. Instead, the usual way of judging seems to depend on (*a*) accessibility, (*b*) amount of possible surgical risk attending removal, (*c*) possible numerical incidence of the organ involved, and (*d*) degree of organization of the specialty concerned in the diagnosis.

(*a*) *Accessibility.* The more accessible the organ the more frequently it is examined and treated. Thus, the teeth lead, with the nasopharyngeal foci following, and the abdominal organs last.

(*b*) *Amount of possible surgical risk attending removal.* The less the surgical risk, the more frequent the removal, and the more voluminous the reports. The teeth again lead in this respect, the nasopharyngeal foci following, and the abdominal organs last.

(*c*) *Possible numerical incidence of the organ involved.* The teeth are again leaders, as there are 32 possible foci in the alveolo-dental region, 10 major sinuses, 2 tonsils, 1 gall bladder, etc.

(*d*) *Degree of organization of the specialty concerned.* The better a specialty is organized, the greater the number of its contributions

to the literature and the wider it is known. Here again teeth lead, since dentistry is the best organized of the healing arts, while nose and throat, abdominal and genito-urinary surgery follow in close sequence. Very often one sees an appendix removed for the relief of chronic spastic constipation in a patient with an anal fissure and hemorrhoids, because they are regarded by the abdominal surgeon as effects rather than causes, when in reality they may be the cause. The lack of prominence of these lesions in discussions on constipation, as contrasted with the apparent importance of the appendix, might be due to the relatively high degree of organization enjoyed by abdominal surgery as compared to rectal.

At the present time, there is very little definite information concerning the disease-producing possibilities of any focus because the method of determining this has been influenced to some extent by the four factors just discussed. However, a brief review of the more common foci may be of some help in this respect.

The *tonsils*, because of their location, blood supply, glandular structure and activity during swallowing, are important foci. They are located in the anterior portion of the pharyngeal area where they are easily accessible to the oral bacteria. Every common cold causes some inflammation in them and, since they are composed of lymphoid tissue, they are very likely to become diseased in the exercise of their function. In modern life with its heating systems and irritation from smoke and other agencies, the tonsils become foci of infection early in life. They have a rich capillary supply and, unless the epithelium which lines the crypts is intact, the systematic squeezing of the tonsil by swallowing forces infectious material into the substance of the tonsil and into the blood stream. Chronic tonsillar inflammation extends to the rest of the lymphoid tissue in the pharynx and causes trouble in the middle ear, upper bronchial tree, and bronchial lymphatics.

The *accessory nasal sinuses*, because of the extensive surface and the ease with which a pan-sinusitis may develop from a simple one, are also important. Chronic sinus disease very frequently causes or aggravates inflammatory diseases in the pharynx, middle ear, larynx and trachea in the same manner as chronic tonsillitis. Pus coming from these regions may reach the stomach and duodenum by swallow-

ing, which undoubtedly favors the occurrence of inflammatory conditions in these organs.

The *teeth* must be considered from the standpoint of periodontoclasia or pyorrhea and periapical disease. I consider the former more important as a focus of infection because of the richness of the bacterial flora and the wide surface exposure, in spite of the fact that it also has free drainage. Surface exposure is very important in judging the virulence of a focus, because a large area has more blood vessels and lymphatics in contact with the infection, which facilitates its entrance into the circulation in sufficient amount to produce disease. Radiographic studies of periapical lesions do not substantiate the opinion that periapical infections are under pressure and are therefore being "forced" into the system. Bone is very sensitive to pressure and literally melts away before any force such as is exerted by a growing soft tissue, as a pericementoma or chronic alveolo-dental abscess. Treatment of periodontoclasia does not give as spectacular results as extraction because it is not attended by the same degree of tissue trauma and, therefore, does not have the possibility of inducing a similar amount of non-specific protein reaction. This apparently increased therapeutic effect has placed unnecessary emphasis upon the periapical lesion as a focus of infection, and this has led to the extraction of many pulpless teeth that had no demonstrable pathological changes.

The *respiratory system* may be the seat of chronic infection such as bronchitis, bronchiectasis, lung abscesses, and parenchymatous lesions. However, it is rarely considered as a focus of infection. The production of one-half to one cupful of pus a day cannot be disregarded, especially since many of these cases suffer from joint disturbances.

In the abdomen there are three commonly named foci, if they can be called such; namely, the *colon*, *gall bladder*, and *appendix*. The colon is probably not the cause of symptoms of malaise, headaches and vague pains. If it does act as a focus of infection, it is only in cases of diverticulitis, ulcerative colitis, and parasitic infestation. Rarely does one find mentioned such foci as *infected hemorrhoids*, *fistulae*, and *ischio-rectal abscesses*. The anal region is just as capable of causing a focal infection as any other part, because it has a large surface exposure and an unfavorable venous drainage.

The *gall bladder* may be a primary or secondary focus. In 11 percent

of the bodies coming to autopsy, it shows evidence of marked pathological conditions, such as stones, without any sign of trouble during life. This can be cited as a true instance of a focus of infection being latent and never producing a focal infection. Wilkie (55) says that the frequency with which this organ is diseased without producing symptoms leads to its contempt. It is a common clinical observation that its removal gives very satisfactory results in functional gastro-intestinal disease even though, frequently, the amount of pathological change in the gall bladder is so slight as to be wholly out of proportion to the improvement experienced. Judd (24) believes that in these cases it is due to a better condition of biliary drainage from the operation, rather than its influence upon the gastro-intestinal tract as a focus of infection.

In the genito-urinary apparatus, the *cervix* and *prostate* are very often the source of infection in the female and male, respectively, with the *tubes* and *seminal vesicles* next in importance. In 100 unselected cases in my office, composed of 50 males and females: 6 males, or 12 percent, were found to have 25 to 50 percent of pus in the prostatic secretion; and 11 females, or 22 percent, had chronic cervicitis. In a purely gynecologic practice the percentage of women with this condition is from 70 to 80 percent, according to Strachan (53). In genito-urinary practice the percentage of men with chronic non-venereal prostatitis is from 65–70 percent. In spite of these figures, it is only rarely that these organs receive primary consideration as foci. *Chronic pyelitis* is another focus which is rarely mentioned.

3. Clinical data

Under the heading of clinical facts, I wish to take up a few of the more important diseases that are regarded as being results of focal infection.

a. Pain. Although pain is not a disease, but rather a sign of disturbed physiology, it must be discussed here because very often it is regarded as a clinical entity. Pain has certain general characteristics, which suggest the procedure necessary to the diagnosis of the cause or help to identify its nature; as for instance, the crushing pain of ischaemia, the constricting pain of colic, the darting pain of neuralgia, and the constant dull pain with acute exacerbations of inflammatory

conditions. In spite of this well established information, many individuals assume that pain is a definite sign of focal infection, and foci of infection are removed for pain in syphilitic disease of the spinal cord, angina pectoris, migraine, etc.

b. Rheumatic diseases. I have found it useful to consider all diseases of inflammatory nature, for which no specific agency has yet been demonstrated, as "rheumatic diseases." This idea was derived from an article which was read some years ago but I have been unable to recall the source. This group includes arthritis, myositis, osteoarthritis, fibrositis, neuritis, iritis, rheumatic fever and its sequelae, etc., and what is said of one is true of the rest. This class of disturbance has been prominent in the bedside data supporting the hypothesis that focal infection is the cause of certain diseases. It has been my experience that, with the exception of rheumatic fever and its sequelae, the improvement resulting from the removal of foci of infection has been more consistent here than in any other group of systemic disturbances. In a series of 200 cases of inflammatory disease that I studied in 1921, 4 percent, or 8 cases, were cured, and 58 percent, or 116 cases, received temporary relief from just the extraction of teeth. Richards (43) in 104 patients with arthritis found streptococcus viridans in the following distribution: teeth, in 50; tonsils, in 40; sinuses, in 11; prostate, in 2; pyelitis and salpingitis, each in 1. Cecil and Archer (8), in a study of 200 cases of arthritis, found that 82 percent were cured or definitely improved by the removal of foci of infection early in the disease. Crouter (40), studying the same disease from a metabolic angle, reported that 60 percent of the patients had lowered blood-sugar tolerance, which returned to normal after the foci had been eliminated.

These figures corroborate the importance of focal infection clinically, but laboratory data reflect doubt that the improvement is due to the removal of the specific cause of the trouble. Holman (21) stated that in rheumatic fever there is a more or less complete upset of the capillary system, seen most prominently in regions having a poor blood supply, such as the joints and endocardium. As was stated before, in the discussion on the incidence of myositis, these tissues are very apt to be disturbed by other factors such as exposure, fatigue, improper diet or menopause, as well as by infection. It is, therefore, reasonable

to believe that, therapeutically, they may be affected as easily by any measure that restores the circulatory integrity of the capillary system for a sufficient length of time to enable it to reëstablish the necessary balance between the blood and the tissues. This is further evidenced by the results from non-specific protein therapy. During this year I have had three cases of proliferative arthritis, with marked gingival and tonsillar infection, two of which also had several teeth with periapical infections. All were treated with injections of typhoid vaccine, without removal of any of the infectious foci, and relief from all symptoms occurred at once; and, since June, there has been no recurrence. Although I do not mean to advocate non-specific protein therapy as a substitute for removal of foci, this may suggest the mechanism by which this removal produces the beneficial effects; namely, that the tissue injury, associated with the operation, liberates foreign proteins, which act as a sort of autogenous non-specific stimulant. Since the diseases in which non-specific therapy is beneficial are practically identical with those in which the removal of foci of infection also produces good results, the temptation is strong to believe that the relief is not due to the elimination of the specific cause in these rheumatic diseases, but to the action of an antigen, like a foreign protein, coming from the injured tissue.

Further evidence that these rheumatic affections may be the result of structural inadequacies or metabolic defects, rather than the expression of a specific agency, is the fact that improvement or cure often follows simple procedures, such as change of climate, rest, physiotherapy, mild attacks of fever, and even psycho-therapy.

c. Gastric and duodenal ulcers. During the last fifteen years focal infection has received considerable mention as a cause of gastric and duodenal ulcer. Rosenow (46) and Haden and Bohan (20) believe that these diseases are the result of elective localization by streptococci resident in dental and tonsillar foci. The gall bladder and appendix are also frequently mentioned as causative foci, and clinical cures have been reported after the removal of these organs. In the laboratory these views have not been consistently verified. C. E. Robertson, pathologist at the Mayo Clinic, in a lecture before the Los Angeles Clinico-Pathological Society this year, stated that in his experience, he was not convinced that ulcers are due to focal infection. McMeans

(32) showed that the experimental ulcers produced by the injection of bacteria are not identical with the initial stage of ulcers in man.

Laboratory proof that peptic ulcers are the direct result of foci of infection is further challenged by the fact that certain laboratory animals are prone to develop these conditions under experimental environment. Rosenau and Anderson (45) found lesions in the stomachs of 66 percent of 2882 guinea pigs dying soon after injections of diphtheria toxins. If those that lived two or three days are included in this list, then the proportion was 77 percent. Carlson (6) reported that 75 percent of the dogs used in experimental parathyroid tetany showed ulcers, but the cats did not. He also stated that the dog is the most prone of all experimental animals to develop these lesions without experimental interference. Friedman (18) was able to produce ulcers and erosions in the duodenum of rabbits by injection of adrenalin and, in the stomach, by pilocarpine. Warren and Whipple(54) produced ulcers with the x-ray, and it is well known that extensive superficial burns often produce duodenal ulcers. Balint (3) found an abnormally high hydrogen-ion concentration in the blood of persons having ulcers, and recommended the alkalinization of the entire body in the treatment of ulcers of all kinds. Shapiro and Ivy (48), in studying the rôle of anaphylaxis, produced acute ulcers in rabbits and dogs, but the rabbit was more prone to skin reactions. So far as I know, none of the work on experimental ulcers with microörganisms gives evidence that this susceptibility of laboratory animals has ever been considered.

In late years it has been noted that the number of duodenal ulcers exceed the gastric. It may be suggested that this is related to the increased strain of modern life, with its attendant emotional load, which causes a greater amount of fatigue. Continued fatigue from emotional sources tends to produce symptoms which suggest spasm in the smooth muscle systems, such as the respiratory, vascular, gastro-intestinal, genito-urinary, and pilomotor. Duke (15), quoting Zinsser, showed that the most marked effect of anaphylaxis is upon the smooth-muscle system which has the greatest development in an animal and causes death with that system in spasm. It is probable that a well developed gastro-intestinal system in the human may show the effects of this type of fatigue by spasm, which if persistent may derange the circulation of the area, lower its vitality, and produce ulcerations of

stomach and duodenum; while, in another, a highly developed vascular system may respond in a similar manner and invite essential hypertension, or, in women with powerful reproductive musculature, dysmenorrhea may result. I have observed that one of the effects of tobacco is to dull the normal sense of fatigue and thus enable the individual to work beyond the point of exhaustion. Such persons are prone to symptoms and x-ray findings of spasm of pylorus and duodenum, which promptly disappears when smoking is decreased or stopped. The treatment of peptic ulcers advocated by Sippy (50) bears out the principle of spasm, because it is essentially a means of mental and physical rest with the induction of rhythmical peristaltic action by frequent feedings and alkalies.

Therefore, focal infection cannot be considered as the primary etiological factor in gastric and duodenal ulcers. However, it undoubtedly plays a secondary rôle by inducing subsequent infection, after the ulceration has been established, through the swallowing of pus or by lowering the general body tone. Infection of the gall bladder, by interfering with the normal biliary and liver functions, may influence these ulcers through disturbance of digestion or reflex irritation that favors spasm.

d. Gall-bladder disease. Phillips (41) reported that cholecystitis and cholelithiasis produced gastro-intestinal symptoms in 82.3 percent of his series of cases, which gives the gall bladder considerable importance as a focus of infection. However, the statement that cholecystic disease is always an expression of focal infection is difficult to prove. Normally, the gall bladder is sterile, owing to the bactericidal properties of the bile. This property is effective only against certain organisms, while others are immune. According to the Fergusons (17), during bacteremias, such as typhoid, pneumonia, or septicemia, organisms are carried to the gall bladder by means of the hepatic artery, while certain gastro-intestinal bacteria reach it through the portal system, so that bacteria occur often in the gall bladder and yet cause no infection. However, Phillips (41) believes that the presence of certain factors such as pregnancy, injury to the gall bladder, and infrequent emptying, together with the presence of organisms, tends to produce cholecystitis.

To prove that bacteria from oral or other foci are a frequent cause

of cholecystic disease is difficult; first, because streptococci are the organisms most frequently implicated and, in this location, they are very hard to identify. Secondly, it is not probable that small numbers of organisms from the teeth or other distant places can be transported by arterial blood without suffering a decreased virulence. Thirdly, the fact that the bactericidal properties of bile, in any case, may be quite effective against one organism and impotent against another. Stone (52) showed that rabbit bile killed all strains of streptococcus pyogenes. Meyer and Löwenburg (34) found the enterococcus favored the gall bladder. This organism lives in the gastro-intestinal tract and is very frequently confused with other strains. Undoubtedly, some of the strains from the distant foci that produce cholecystitis, which are said to be recovered from the gall bladder, are in reality strains of the enterococcus. This is further indicated by the work of Stone who found that the bile from cow, pig, dog, sheep, cat, guinea pig, turtle, and man, had no effect upon the streptococcus pyogenes. Since this microörganism is often used in the rabbit, whose bile in small quantities kills it, then any recovered organism is not the same but rather a native germ. Therefore, reports that limit their descriptions of the microörganisms found in the gall bladder as a result of the experiment, to short-or long-chain streptococci, are definitely misleading.

It is plain that one cannot definitely state that foci of infection are a necessary part of gall-bladder disease, yet this view is the most frequently enunciated idea concerning its etiology in medical practice today.

e. Pancreas and diabetes. Studies in blood-sugar tolerance, and its relation to certain skin disturbances such as dermatitis, erysipelas, and furunculosis, show that microörganisms, in the presence of an increased blood sugar, take a prominent part in the development of these diseases. Ayres (2) found high blood-sugar-tolerance values in the eczema group and, in his series, 12.7 percent had 200 mgs. or more.

McLester (31) noted the effect of infections in diabetes. It is well known that a common cold causes a rise in blood sugar and sometimes may precipitate a coma. Joslin (23) does not believe that infections are important since he could find them in only 15 out of 400 patients.

f. Heart and endocarditis. In recent years the heart has received a great deal of attention owing to the apprehensiveness of the laity, the

better diagnostic skill of physicians, and improvement in diagnostic methods. The most important single cause of heart disease is rheumatic fever, because it occurs in childhood and leaves this organ crippled in nearly one half of its victims. According to Church's (10) series, 494 out of 889 cases showed evidence of old or recent endocarditis. A great deal of work has been done in trying to explain the etiology of this disease upon a bacteriological basis, although up to the present time no organism has found general acceptance as a causative factor. The majority of workers favor some form of streptococcus, especially the viridans. Because of the frequency of its occurrence in the naso-pharynx and teeth, it is quite natural to assume that the relation between these foci and cardiac disease is very important. The clinical evidence favoring this view is far more convincing than the laboratory. Robey and Freedman (44) recommend tonsillectomy as the most effective measure against rheumatic fever. Numerous corroborations of this opinion appear in the literature. Christian (9) says that in cases where the tonsils are repeatedly infected and enlarged, with the upper cervical lymph nodes palpable, removal is indicated, but does not think removal in every case is justifiable.

Attempts to prove, by means of laboratory experiments, that focal infection is a cause of heart disease, are beset with numerous difficulties. Bayne-Jones (4) was able to show that endocarditis is an embolic disease by demonstrating the capillary bed along the line of closure of the valves of the human heart, and that the distribution of the vessels and the blood supply varies within wide limits at different ages and in different individuals. It has been said that the capillary bed is very susceptible to any alteration in the body brought on by fatigue, deficient diet, and exposure, which may affect the blood supply to such an extent that a resident microörganism may become effectually implanted upon the valve. Holman (21) stated that cardiac disease is quite easy to produce in animals experimentally, and that sufficient attention has not been paid to the ages of the animals used or to the peculiarity of distribution of the blood vessels in the different species. This writer undoubtedly wishes to call attention to the fact that the younger the animal, the more unstable the capillary distribution and the greater the incidence of the experimental disease; and also that the incidence may be greater in a certain type of animal. Miller (35)

found that 60 percent of his healthy adult rabbits had interstitial myocarditis without presenting any evidence of an infection. Undoubtedly, such a lesion makes the heart more susceptible, apart from any specificity of the organism itself. In spite of this well-known fact concerning rabbits, a considerable amount of work on the bacteriology of endocarditis by recognized workers has been done on these animals, and no provision has been made for this source of error.

Rheumatic and senile heart disease are both intimately connected with the capillary distribution since, in the former, the system may not be fully developed, while in the latter it is gradually failing. Even when rheumatic cardiac disease occurs in adults, or senile changes in the relatively young, it is not hard to show that in the adult the capillary bed is defective, and that obliteration of the capillaries can take place in younger persons owing to premature degenerative changes. As an example of a focus of infection producing a focal infection, one may cite the heart with bacterial endocarditis distributing septic emboli to the different organs.

These differences in anatomical and individual susceptibilities are of sufficient importance to enable one to question the statement that all foci should be removed because of the possibility that they may produce cardiac disease. They indicate that one has time to consider carefully the factors suggested by Christian before advising the removal of a merely suspicious area that cannot positively be proven to be a focus of infection.

g. Nephritis. Starling and Verney (51) believe that kidney function is a process of mechanical filtration and absorption, whereby these organs free the arterial blood from certain soluble products. During this activity bacteria may be arrested in the glomerulus. The kidneys are essentially vascular organs with an extensive capillary bed and, therefore, are subject to the same environmental influences that disturb capillary function in such other areas as joints, fibrous tissue, and endocardium; with the exception that the kidneys, being deeply located in the body and surrounded by a thick layer of fat, probably require a greater degree of disturbance before they show the effects.

Rosenow and Meisser (47) performed a unique experiment which, they believed, satisfied all the objections to the technique in favor of

elective localization. They used a strain of staphylococcus taken from the nasal mucous membrane of a patient suffering from sinusitis and recurrent attacks of acute nephritis, and inoculated dogs through the teeth by opening into the root canals and depositing therein a heavy suspension of bacteria. Several months later radiographs showed typical shadows of periapical infection. The kidneys were found to have the pathological changes characteristic of acute nephritis. In spite of the care taken, this cannot be said to be similar to the devitalized-tooth focus in man; they merely used the root canal instead of a vein as the avenue of inoculation. No tables were given for this experiment covering the number of incidences of infection in other organs, but those showing the distribution of this germ in rabbits have high incidences similar to those in the other organs discussed above.

There are many reasons why experimental nephritis produced by bacterial inoculations must be carefully analyzed before drawing conclusions. First, the unyielding nature of Bowman's capsule and the labyrinthine course of the vessels of the glomerulus make this part of the kidney more vulnerable to the inflammatory changes. Second, Longcope's (27) studies with repeated protein injections show that it is susceptible to anaphylactic reactions. Third, the effects of cold and diet: Seigel (49) produced nephritis in dogs by putting their feet in cold water. Evans and Risley (16) pointed out the injurious effects of a high-protein diet, although Drummond, Crowden and Hill (Addis, 1) were unable to confirm these findings after feeding rats for 100 days on 83 percent casein diet. Fourth, Leiter (26) found spontaneous nephritis in 40 percent of his adult control rabbits. Ophüls and McCoy (38) made the same observation on wild rats. One or more of these factors are always present, so that the addition of another one, in the shape of a bacterial inoculation, may get the result the worker is looking for.

However, most observers agree that acute and chronic kidney diseases either begin and continue as infections, or are the result of a secondary infection implanted upon a weakened organ. Therefore infection, whether local, focal or general, becomes a leading factor in the study, etiology, and treatment of nephritis. Although the laboratory favors the removal of foci of infection, the bedside data are not favorable because the results obtained are rarely satisfactory. It

might be suggested that chronic nephritis is an insidious disease and probably develops slowly, so that when it has produced sufficient signs to permit diagnosis, it is so advanced that no remedial measure can influence it.

h. Eye diseases. Ophthalmologists have been very enthusiastic regarding the relationship between infections of the teeth, tonsils, and sinuses, and the various inflammations of the eye. The relief and cure, which often take place within a very short time after the removal of foci of infection in iritis, keratitis, conjunctivitis, and corneal ulcer, form one of the most brilliant chapters in the literature of focal infection and ophthalmology. It has been my experience that foci located in the head seem to have a much more intimate influence on these inflammatory eye diseases than the distant ones such as prostatitis or cholecystitis although, occasionally, abdominal foci such as ulcerative colitis may be of importance. Mills (37) reports a relationship between eye disease and amebiasis.

i. Miscellaneous diseases. Other diseases included in the scope of this discussion are those involving the *nervous system*, such as poliomyelitis, herpes zoster, epidemic encephalitis; the *vascular system*, such as thrombo-angiitis obliterans; the various *chronic skin manifestations*, such as acne and pustular eczemas; and other disturbances in the organs already mentioned, such as kidney stones, ulcerative colitis, and hepatitis. However, to canvass these would only prolong the discussion without clarifying the subject, since the same points apply to them as to those already mentioned.

IV. FOCAL INFECTION NOT A SPECIFIC AGENCY

From the evidence already presented, focal infection cannot be regarded as a specific agency bearing the same relationship to disease that the typhoid bacillus bears to typhoid fever. Neither is it a negligible factor as Putti (42) the renowned Italian orthopedic surgeon believes. Somewhere between these two views lies the true worth of this principle, which will remain in doubt so long as there is any uncertainty as to how a focus of infection affects the system.

I believe that this search for evidence of specificity has been misleading, and has tended to give focal infection a property which it does not possess. Perhaps the mode of action is basic, similar to that which

fatigue, defective nutrition or exposure to heat, cold, or light, produce upon the body, rather than directly upon organs or tissues. Recent studies in these subjects indicate that this is not improbable.

I shall take up a few of the significant facts in these various fields under the headings of (1) diet, (2) effect of heat, cold, and light; and (3) fatigue.

1. Diet

It was not until the vitamins were studied that the subject of defective nutrition with respect to disease was fully appreciated. Recent data seem to show that deficiency in vitamins increases susceptibility to infections. Cramer (13) believed that certain organisms may be non-pathogenic on one diet and pathogenic on another, depending on the concentrations of vitamins A and B. McCarrison (29) stated that his polyneuritic birds (vitamin-B deficiency) were more susceptible to blood-stream infections with Bacillus suipestifer, Bacillus pyocyaneus, and another unidentified organism. McCollum (28) called attention to the fact that the lymphopenia and atrophy of lymphoid tissue throughout the body in vitamin-B deficiency is similar to that produced by x-ray exposure. Since these tissues are one of the defensive agencies against bacteria, infection would naturally follow. Lange (25) showed that deficient salt and protein content in the diet increased the susceptibility of the rat to bovine tuberculosis, while Coulaud (12) stated that the course of experimental tuberculosis is greatly hastened in the guinea pig by a deficiency in vitamin C. Green and Mellanby (19) fed 93 rats a diet deficient in vitamin A. Postmortem examination showed 91 to have infections located as follows: abscesses involving the salivary gland, base of the tongue and cervical lymphatics in 72 percent; infections in the bladder and kidney in 44 percent; gastro-intestinal tract infections in 21 percent; middle ear or nasal sinuses in 20 percent; septic broncho-pneumonia in 9 percent. Holman and Miller (21) found that a high-carbohydrate diet in rabbits and guinea pigs made them all more susceptible to abscesses from small inoculations of staphylococcus aureus, which did not affect the control animals. Deficiency in vitamin A produced urinary calculi in rats according to Osborne and Mendel (39). A diet lacking in vitamin C was observed by Cohen and Mendel (11) to produce tender, swollen, and painful joints in growing animals.

2. Effect of heat, cold, and light

The effects of heat, cold, and light depend for the most part upon the changes produced in the capillary system. Wright (56), quoting the experiments by Lewis on the action of histamine, points out how the capillaries take part. Histamine produces a paralysis of the arterioles and capillaries, which prevents the return of the venous blood from the part involved. Heavy stroking or injury to the skin liberates a histamine-like substance having the identical reaction. Exposure of the skin to a temperature of from 40 to 45°C. causes redness and dilation of the capillaries owing to the combined influences of (*a*) liberation of histamine-like substance; (*b*) direct action of the temperature on the capillaries; (*c*) reflex dilation of the larger vessels causing increased blood-flow through the skin. Higher temperature causes the typical flare, wheal, and blister, owing to the liberation in greater amounts of the histamine-like substance.

Cold also affects the capillaries. It produces a contraction, with depression of the oxidative function. But it is only when freezing takes place that the histamine-like substance is set free. The application of ice in inflammations produces a counter irritation that relieves the disease area.

Light therapy, in the form of rays from the sun, carbon arc, heat, and mercury vapor lamps, owes its beneficial effects to the erythema produced, which is also a response to the liberation of the histamine-like substance. These agencies produce a more lasting effect on the capillaries than heat or cold and, for several months after the exposure, the redness from stroking the skin is more marked.

Heat, in the ordinary form, does not have the therapeutic value that fever does. It has been noted for some time that chronic disturbances were benefitted by an attack of intercurrent febrile disease. Non-specific protein therapy depends upon the fever which is produced. The injections into the system of milk, peptones, dead typhoid, and other bacilli, and the use of the malarial parasite in cerebro-spinal syphilis, are other ways of causing the reaction. The heat of fever must activate all the capillaries of the body in the same manner as when applied locally.

Non-specific protein therapy has been used in a number of diseases, and the percentages of cures, together with the rapidity with which the

symptoms are relieved, are almost identical with the results obtained from the elimination of foci of infection. Miller (36) reported freedom from pain in 70 percent of cases of acute arthritis, and in 35 percent the improvement was permanent. His results in chronic arthritis were not so favorable, but Yoeman (57) obtained cures in 50 percent of this group. Since arthritis is representative of the rheumatic group, these figures indicate the possibilities in the rest of these diseases. Peptic ulcer was treated in the same manner by Miller, and he reported striking improvement from two injections of dead typhoid bacilli. Howard (22) treated 66 cases of eye diseases, composed of keratitis, conjunctivitis, iritis, uveitis, corneal ulcers, and secondary glaucoma, with typhoid-paratyphoid injections alone, and obtained marked improvement in 62 cases or 94 percent. These figures are almost identical with those reported after the removal of foci of infection. Chronic coughs and other respiratory diseases have been treated with vaccines and serums, but with indifferent results, and the success from the removal of foci is about the same. Allen, Brown and Mahorner (55) highly recommend the injection of non-specific proteins in the treatment of the pain in thrombo-angiitis obliterans.

3. Fatigue

Fatigue is caused by long continued muscular movements. Associated with them is an increased demand for blood, accompanied by a rapid flow through the lungs, and imperfect oxygenation results. Wright (56) stated that, when the blood supplying the heart has even a slight diminution of oxygen, the efficiency of the organ becomes so impaired as to render the circulation inadequate for the needs of the body. This condition, together with a slight increase in the pH concentration, depresses the brain and produces a nervous irritation causing a vasomotor reflex, and capillary upset ensues. Thus the mechanism of lowered resistance favorable for the onset of an infection is at hand.

Since the diseases that follow the effects of defective nutrition, fatigue, and exposure to heat, cold, and light, are very similar to those said to be the result of focal infection, it is apparent that focal infection is not the specific cause of any pathological expression, but rather a general influence of endogenous character that deranges the

defensive mechanism and permits the native bacteria, usually streptococci, to produce an infection. This was clearly illustrated in Green and Mellanby's experiment (19) where 91 of the 93 rats, fed on a diet deficient in vitamin A, developed pyogenic infections. It is plain, from these data, that the importance of focal infection cannot be measured by a bacteriological standard such as Koch's postulates, because it depends upon that subtle and variable property, the ability of an individual to maintain a physical and chemical balance. Therefore, the power of focal infection to produce diseases is no greater than that exercised by agencies such as faulty nutrition, fatigue, and exposure to heat, light, or cold.

V. GENERAL CONCLUSIONS

These studies tend to disprove the specific relationship between a focus of infection and a disease. Therefore, it is suggested that this doctrine of specificity be abandoned in favor of the broader concept that it is a basic influence affecting the whole body. That the benefits derived from the removal of foci of infection are not due to the elimination of a specific agency, as is believed, but to the introduction into the body of an autogenous substance, which produces the same effect as a foreign protein, is evidenced by the histamine reactions of Lewis; the rapidity of relief from the symptoms, sometimes said to occur within an hour; and the striking similarity not only between the diseases benefited by non-specific protein on the one hand and removal of foci on the other, but also in the percentages of the results themselves. Thus, the removal of all foci merely as a routine measure is not justified, because not all foci are active, and it is necessary to prove that they are actually the cause of the focal infection. Another point, rarely considered, is that every inflammation is associated with protective and regenerative activities, making it possible for a focus to undergo spontaneous cure. It must also be remembered that there are many hidden and inaccessible areas, the importance of which is never stressed because they are not in plain view, but which may be responsible for the trouble. Therefore, the removal of foci of infection should be carried on in proper relationship to the rest of the therapeutic program, and in accordance with definite and practical indications.

VI. SUMMARY

1. Focal infection concerns the dentist more than the physician.

2. A focus of infection may remain inactive and never produce a focal infection. Therefore, the terms "focus of infection" and "focal infection" are not synonymous.

3. Clinical and laboratory data do not always agree on the diseases caused by foci of infection.

4. The four theories of focal infection do not aid in determining its mechanism.

5. The importance of a focus of infection has not been determined in any case because it depends upon surface exposure, blood supply, structure and functional activity, and not upon accessibility, surgical risk, numerical incidence, and organization of the specialty concerned.

6. Laboratory data do not verify clinical data with respect to the reasons for the results obtained from the elimination of foci of infection.

7. Recent studies in nutrition, and the effects of heat, light, cold, and fatigue, show the similarity between diseases resulting from these influences and those said to be due to focal infection.

8. Focal infection is not a specific disease-producing agency, but a general influence that lowers the body resistance and thus permits native bacteria to cause infection.

9. The beneficial effects derived from the removal of foci seem to be due to an autogenous non-specific protein reaction, which develops from the tissue injured during the operation and not as a result of the eradication of a direct causative factor.

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DISCUSSION

Nellis B. Foster, M.D. (*New York City*): I regard it a privilege to discuss this paper because, quite candidly, it is one of the best presentations of the subject I have ever heard. It is a judicial presentation. Those of you who came here feeling rather strongly in favor of the argument for focal infection may not feel it is a judicial presentation, because, I should think, your theories are pretty well shot to pieces. The doctrine of focal infection is an interesting one. I have seen it develop and reach its apex. From the medical point of view at least, it is now definitely on the wane. The person who gave focal infection its great impetus was probably Frank Billings of Chicago, and he, having repented, has changed his point of view.

I cannot say very much in discussing the paper because I am not able to dissent in any essential particular from the points of view presented. As was pretty well brought out, the basis on which the doctrine rests, or did rest, is chiefly clinical. Of course, in a large way, one knows that such things as a focus of infection and a focal infection can exist. Every one of you knows somebody who has contracted a little cut on his finger and has had to lose his axillary glands because he developed a septicemia. Those things do occur; an infection that is localized becomes a systemic infection. That is a well-known thing. But as was brought out, this does not explain the reason why a focus of infection in a tonsil, or tooth or somewhere else, gives rise to one of the diseases the etiology of which is still in dispute. This clinical evidence, since the bacteriological and laboratory evidence is misleading, does not stand up very well under examination.

Now, in the very nature of enthusiasts, what are we given as evidence?

The persons who write the papers are those who have the "successes." Frankly, do you as conscientiously, carefully, and enthusiastically report your failures as you do your successes? No, you do not. You keep still about them. It is human nature. But, of course, for the advancement of the scientific side of the subject, we ought to know all those who, having attempted this method, had nothing but failure as the result. It is undoubtedly true that one does see improvement following the removal of foci of infection in certain individuals. The fault is that the series of cases are usually too small from a statistician's point of view. Two hundred cases is not enough to prove anything, unless somebody else can continue that series and accumulate thousands. A scientific therapeutist, a century ago, said it took ten thousand cases to prove a principle. Dr. Osler used to say that the worst thing about quackery was that it often cures. I am fond of telling about an early experience at the New York Hospital, some years ago, in the treatment of a disease that has a 98 or 99 per cent mortality; namely, ulcerative endocarditis, known as malignant endocarditis. It is practically always a fatal disease. Dr. Elser, the pathologist at the New York Hospital about twenty or twenty-five years ago, hit upon a new principle in bacteriology and we attempted to test it therapeutically. In order to do so, we thought that the best class of cases would be that in which the mortality was highest. We always had cases of ulcerative endocarditis in the wards. They practically always died. We treated four cases on this new principle, and the first three got well. After the first two had gotten well, to say we were enthusiastic would be putting it very mildly. I think that after that first three we did not see a single case get well, and after a couple of years, we entirely abandoned the method. Often, for some reason unknown to statisticians or to scientific medicine, that is the experience with new methods. They are apt to cure for awhile. But the point that I wish to make is that because a few patients were improved (very few have claimed to be definitely cured of chronic arthritis), the correctness of the theoretical basis on which they were treated is not verified. What are we going to do about it?

It is undoubtedly true that a streptococcus infection does not improve a poor condition a particle. We say of erysipelas that it kills only two classes of patients, the very old and the very young. Why? Because they are in the weakest stage of their life period. I think we must take the attitude that, while we are exceedingly skeptical about the theory as it was originally given to us—that is, we are very doubtful whether any disease is caused primarily by a focus of infection—yet it is sometimes worth experimenting to find whether the removal of a focus, provided we can be sure of it, will

so improve that individual's health that he will throw off whatever poisons there are in his system. Proceeding thus, you would perhaps change his diet, or the scene of his activity, not because you are sure it will do any good, but because you think it may. From this point of view I think we can still handle a few infections in an intelligent and a scientific way. However, do not conclude that we can take the attitude—like many in the past—of advising the removal of foci of infection as a procedure to bring about a definite cure. The disappointments and humiliation to patient and physician are too great. We must be frank. If the patient is willing to share his part of the burden of responsibility, then, if he has the right to decide, it is worth while to attempt this experiment.

Sigmund W. A. Franken, D.D.S. (New York City): Those of you who have for years followed Dr. Pollia's outstanding work, both written and oral, have anticipated his very interesting and enlightening paper, with its quintessence of over fifty references. It is indeed a great temptation to go over each point and either agree or disagree giving appropriate reasons, but such a course would necessitate another paper rather than a discussion. I will, therefore, immediately refer to his conclusions, and revert to his text only for the sake of clarity.

The first of the conclusions states that focal infection concerns the dentist more than the physician. The one it concerns most, however, to my way of thinking, is the patient. Then comes the academic discussion of physician and dentist. Even if Dr. Pollia meant dental infection, instead of focal infection, it is doubtful whether his conclusion is correct. It is the duty of the internist to diagnose, alleviate, and, if possible, cure the patient. One of the many means at his command is the search for and the removal of any infection. He is entitled to all the aid procurable from the various specialists, such as rhinologist, gynecologist, proctologist, etc., as well as the dentist or, rather, the dental diagnostician. The questions put to these various men should be: First, is there infection present in your particular field? Second, can it be eliminated, and how much of a surgical shock may follow such removal? Then it is up to the internist to decide who should begin. Dr. C. H. Mayo had this in mind several years ago when he stated that dentistry is as closely related to medicine as the specialties of eye, ear, nose and throat, or any of the others.

Very often a patient suffering from arthritis happens to have the hypertrophic type, which, as has been said by Osgood (referring to the Report to the American Committee for the Control of Rheumatism), is a disease of middle age or old age. It is a form of involution. Another patient has an atrophic

arthritis, which is infective in origin. These types are not always easily differentiated. If it is hypertrophic, the removal of the doubtful teeth will not be of much aid, whereas if atrophic, which is infective, much benefit may result. How many dentists are qualified to differentiate such conditions; and besides, what right have they to practise medicine? Focal infection is, therefore, not the province of, nor should it concern, the dentist, or the medical specialists, as much as it does the internist or the general practitioner.

The next fact in the summary, concerning the differentiation between focus of infection and focal infection, has been beautifully explained and should be accepted.

That laboratory data do not verify clinical data with results obtained from elimination of foci of infection, follows, I venture to say, the usual course of medicine applied to therapeusis. For generations, cod-liver oil has been administered, and the laboratories showed simply a high caloric benefit. Not until a few years ago did the accessory food factors, better known as vitamins, have any recognized significance. Yet results were obtained long before this discovery of vitamins, and cod-liver oil was just as beneficial then as it is now. Another such instance is the feeding of liver to patients having pernicious anemia. In short, empirical medicine is still waiting and will wait, in most instances, for the laboratory to catch up with it.

The four theories of the mechanics of focal infection, namely; (1) Rosenow's elective localization, (2) Meyer's organ selective action, (3) anaphylaxis or protein sensitization, and (4) point of lowered resistance, are next discussed. But, as already stated, a very important one, perhaps the most important, was not mentioned; that is "the patient himself." Dr. George Draper has been doing some highly interesting investigating of types of patients susceptible to different diseases. He has worked mainly on the anatomical panel, and claims to have special indications why some individuals are liable to peptic ulcers, others to gall-bladder disturbances, and still others to pernicious anemia. That there are people more susceptible to the streptococcus than others there can be no doubt. Very often it happens that two or more persons are exposed to the same infection, yet one succumbs, while the others are not even disturbed. Only two weeks ago, Dr. Macynty, at the Academy of Medicine's Graduate Fortnight, said that the next important happening for which we are all waiting is the arrival of a great immunologist. There lies the kernel of the problem: why do some become ill and disabled, while others escape with impunity from an infection that appears to be even more virulent.

Dr. Pollia touched upon the mind, and its strange and unknown effect upon the general physiology. That is true. And it is also true that in a family of children under similar stress and influences, one may develop into a manic depressive, another into a schizophrenic, while the remaining one or two might be what is termed normal. This has happened again and again, and I believe the same reason underlies this phenomenon and the one under discussion; namely, the susceptibility of the patient.

The rest of the summary, excepting the last paragraph, deals with the various foci of infection and some of the diseases for which they are reputedly responsible; and, conversely, with several diseases whose etiology is, supposedly, focal infection. The fact that some of the abnormal physiological states can be corrected, as explained in the paper, by change of climate, diet, etc., seems to me to indicate, again, nothing against the theory of focal infection but, rather, the bringing to the fore the fact that this is a question for the internist. When should the patient indulge in a heavy intake of vitamins? When should he take a vacation, or change his diet and climate? When should he take on some physio-therapeutic measures, or even psychoanalysis? And last, when should he have the foci of infection removed? Surely, neither the rhinologist, gastro-enterologist, stomatologist, nor any of the other specialists are in as safe or sane a position to answer as the internist. Ralph Pemberton knew this when he wrote that focal infection, including the dental, should be removed when not contra-indicated by the balance of health or severity of surgical procedure. Also, that a balanced view-point is necessary to determine cause of illness, to achieve improvement, and even to avoid further injury.

Russell Cecil, only a few months ago, emphatically maintained that focal infection has been placed where it belongs, as one of the foundation stones of modern medicine. Mind you, he doesn't claim it to be all of medicine. I believe Dr. Pollia has rightfully challenged our notorious hundred-percenters. The absurd idea of extracting all devitalized teeth in all people, healthy or diseased, old or young, is a flare-back to the ages of fanaticism and ignorance. It is, indeed, a sad prospect for dentistry, if there is no hope for teeth whose pulps have been involved. If this attitude had been permanently adopted by the medical profession, there would today be neither insulin, salvarsan, nor any of the other specific remedies. This searching for cures is the yeast necessary for advancement in the healing arts, and dentistry should not be excluded because of the uncertain present.

As to the final conclusions, relative to a non-specific protein reaction and focal infection, one is reminded of the usual gamut through which a new therapeutic agent must pass. At first, it is derided, then frantically ac-

claimed as a panacea for all ills, and, ultimately, after the usual train of disappointments, the truth is finally established in its relationship to other remedies. This applies as well to specific protein therapy, known also as "shock therapy." At first it had few adherents; then, when general paresis was treated, not unsuccessfully, with malaria, it was taken up with a vengeance; and at present it is in its second stage, where it is being used to excess for all kinds of diseases, as well as in cases of acute and even chronic infections. Later on—let us hope soon—it will settle to its proper position.

If, as Dr. Pollia proposes, the cures after eradication of dental foci are brought about by the liberation of this autogenous, hypothetical, non-specific protein, how does it happen that after the extraction of infected teeth, with the attendant trauma due to broken apices, the patients frequently do not recover from their systemic involvement? As brought out by Haden, Keeling and others, how is it that so many cures are effected after the removal of these remaining apices and residual areas of infection, sometimes years later? How is it that, after tonsillectomies where there are remaining tags, these tags must later be removed before improvement occurs in the patient. What happened to the autogenous specific toxins that had been liberated? Why did they not effect the cure? And again, if the ideal is an autogenous non-specific reaction, why not merely incise a thigh for several inches and close it up again, and then expect a cure of the arthritis?

Dr. Pollia correctly points out that there are so many factors at work in this problem of focal infection that one must be wary of conclusions. He puts forth, very strongly, the preponderant value of the non-specific protein therapy. This I object to for the same reasons that he objects to the other theories. There are other reasons, such as that indicated by Dr. Irons last week at the Academy of Medicine, to the effect that the mechanism of non-specific protein therapy is unknown and, at times, dangerous. Often shock occurs, which sometimes has a fatal result. It is a dangerous therapeutic, indeed! Then again, merely because the use of non-specific therapy aids those suffering from general paresis, is that any reason for discarding salvarsan and mercury for the treatment of syphilis? If this be so, what a misfortune for dentistry! If we reason this idea to an end, what an absurdity do we find. No more asepsis necessary! If a pulpless tooth shows an apical area, shock therapy is all that is required. No more tedious and difficult root-canal surgery or therapeusis—merely some milk injections and the tooth will be sterile.

Theodor Blum, some time ago, spoke of scientific research in focal infection, and said it regards first, the bacteriologist, second, the physician, and

third, the dentist. I would like to modify this by saying that the clinical version concerns: the patient, what he is and how he functions; the internist, to decide the modus operandi; and the dentist and other specialists to decide the presence or absence of infection.

In closing, I would like to add that we should bear in mind the various points Dr. Pollia brought forth, and should mull over them from an academic point of view as to the mechanism involved. But what should be particularly stressed before an audience of dentists is the newer aspect that, clinically, our work is clear; that our particular province in focal infection is a distinctly limited one, and its limitations are self-evident. Briefly, then, we can tabulate as follows: First, to ascertain, if possible, whether focal infection is present. Second, to ascertain whether this infection can be removed. Thirdly, if the answers to 1 and 2 are affirmative, it is then "up to" the internist, after receiving necessary reports, to decide when and which foci, and in what order, the various foci found in the patient should be removed.

Dr. Pollia (in conclusion): I wish to thank Drs. Foster and Franken for their kindness in reading my lengthy manuscript and trying to reach conclusions. In reply to Dr. Franken's statement that I advocate non-specific protein therapy as a *substitute* for the removal of foci of infection, I am going to do an unconventional thing, and ask those members of the audience who understood me in that manner to please raise their hands. [Dr. Pollia paused and as no hands were raised, continued.] I feel relieved since I was afraid that you might have had the impression that Dr. Franken derived. My idea regarding non-specific protein therapy is that curative effects are obtained by its use in the same diseases that are benefited by the removal of foci of infection, and I am using it simply as an argument against the idea of specificity of focal infection as a disease-producing agency. I also want to state that I regard the removal of foci of infection as having a distinct therapeutic value, but I do not believe you thus actually eliminate the specific cause of the pathological expression.

I am a physician and have done a lot of work with dentists, and feel rather proud that the dental profession has "adopted" me. Also I am glad to state that a considerable amount of my so-called success has been due to the support of its members throughout the country. Therefore, I would like to tell you how the situation looked to a physician when the theory of focal infection hit dentistry, because it forms the reason why I believe that focal infection concerns the dentist more than the physician—not from the standpoint of the patient, but from that of the theory and practice of den-

tistry. The main objective ever since the beginning of dental thought has been to save teeth. Along comes the theory of focal infection, and states that under certain conditions the teeth become foci of infection and, therefore, a menace to health. In parenthesis it says, "Therefore some of your traditional operations that you have spent so much time on, such as removing pulps and filling root canals, the making of some types of abutments for bridges, and in many instances the finest kind of dental work, is wrong, because it creates those conditions and is therefore a menace." It is also a rather striking and interesting fact that the quack dentist did not take the same pains with his work and, therefore, the poorer class of patients seemed to be much better off in this respect than those who could afford to have good dentistry, because work done by the so-called charlatan rarely included the removal of pulps.

Without warning this achievement, and one of the most important operations of dentistry, is attacked, carrying the indictment that it is the means of inviting disease and death, and which if true, relegates into the discard a most important dental tradition. Physicians have not helped the dentist in this trouble, but have increased the confusion by arbitrarily taking matters into their own hands, and dictating the removal of teeth and many times hurrying the issue, thereby robbing the dentist of the opportunity for careful systematic observations that would help to orient him on the question. Physicians have failed to accord the dentist in many instances the common courtesy of equal voice in a consultation due to a professional colleague and, further, have failed to appreciate that a dentist is entitled to this courtesy since dentistry is the most highly specialized of the healing arts. I make this claim because seven eighths of the time used in training is spent in studying the mouth, and all of the years of actual professional life are devoted to the teeth and oral tissues. Therefore, all other things being equal, it stands to reason that a dentist knows more about teeth and has a better intuition, if you will permit the term, than any physician.

This brings us up to the present. Until very recently one finds the dental profession split into two groups: those who believed that all diseased teeth should be extracted without exception and regardless of the extent of the pathological process, and those who felt that a number of diseased teeth could be saved by treatment. Many times one felt that the division was based upon the ability of the members of these two groups to extract or to treat, rather than upon actual clinical and pathological factors. Therefore, every dental operation used today is being judged by its possibility of injury or death to the pulp, because we have been led to believe that the operation of pulp removal seriously menaces the health of the tooth and related tissues,

and may result in a focus of infection. Since foci of infection are an important consideration in the study of health and disease, it is obvious that focal infection does exert a controlling influence on every dental procedure, even to the methods of extraction.

I call your attention to a very amusing situation among eminent specialists. Dr. Franken quoted Dr. Osgood on arthritis. He states of this disease, that there are two groups: the hypertrophic arthritis, which he says is not due to infection, and the atrophic, due to infection. I should like to quote Dr. Ely, an eminent bone and joint authority, who states that the hypertrophic form is due to infection, and the atrophic is not.

A word on Dr. Draper's work on constitutional patterns and susceptibility to disease, and its application to focal infection. This is a thought similar to that of the *locus minoris resistentiae*. It is a very fundamental observation, which faces us at every turn like heredity, and our problems had better be approached without letting such factors dishearten us, because our progress must be made in spite of them. More practical, I believe, is the fact that the bacteriology of focal infection is the bacteriology of streptococci, which have a wide distribution in nature and extensive distribution in the animal body. One doesn't need to import outside organisms to produce an infection. All that is needed is to lower the body resistance by any one of the many conditions that influence the circulatory balance of the capillaries, and the native streptococci either residing in the organ or in the body will do the rest.

What is the reason why, referring to Dr. Franken's question, the first operation did not produce the hypothetical non-specific protein substance necessary for cure, whereas the re-operation of the area did? I might state that I do not know, except perhaps that in the first instance not enough tissue was injured to produce those substances or else the body was unable to respond to the stimulus at that time.

Those of you who have heard me lecture before probably recall that I then used the example of the 200 cases of inflammatory disease in defense of conservatism in the removal of dental infection. This evening you see these same 200 cases used as evidence in favor of removal of foci in that group of rheumatic disturbances, and it was pointed out that 116 cases or 58 per cent were temporarily relieved by the procedure. Non-specific protein therapy produces results that are at least no worse, and one must not judge its merits on the cures alone.

Now I want to say something about specificity of drug action. I wonder if there is any such thing as specific drug action where the agency actually produces the curative result by direct action on the causative factors, except

in just a very few instances in anti-toxin therapy? You know Pasteur never accused bacteria of being the cause of anything—he merely believed that they altered environment. It was Koch who developed the four postulates upon which the specificity of bacteria were to be judged, and thus brought the principle of specific cause and effect to the front. I am not so sure that this principle of specificity has not been somewhat of a yoke on medical thought, for it tries to reduce broad fundamental end-results to a simplicity that is impossible because of our scant knowledge. Even such chemotropic specifics as quinine and the arsphenamine group do not depend upon the parasiticidal property alone, but require the coöperation of some unknown agency developed by the body through the influence of the drug for the actual removal of the parasites from the system. Large doses of quinine, the arsphenamines, mercury, and the salicylates are associated with definite disturbance of the capillaries, as indicated by erythemas of various kinds. This may be a hint as to their mode of action; namely, the restoration of balance between the circulatory blood and the terminal tissues. This observation tends to weaken the universal doctrine of specificity; and as knowledge is being added, it is evident that these phenomena often are the result of several factors rather than one alone.

In closing the discussion, the thought I want to leave is that focal infection is not a predominating etiological factor. It is but one of the many conditions that may affect health, and has no greater influence than defective nutrition, exposure, fatigue, or emotional strain.

STUDIES, IN THE RAT, OF SUSCEPTIBILITY TO DENTAL CARIES I. BACTERIOLOGICAL AND NUTRITIONAL FACTORS[1]

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I. INTRODUCTION

The present report deals with the first of a projected series of investigations aimed to determine those factors in the etiology of dental caries that relate to the problem of susceptibility. Among the many possible factors involved in this problem, four, which have been given prominence by recent investigations, are dealt with in this study: the possibilities that (*a*) the presence or absence of lactobacilli may determine the incidence or absence of caries (14, 15, 22, 1, 2, 3); (*b*) the character or amount of carbohydrate in the diet may affect the incidence of caries directly (24), or determine the presence or absence of

[1] This work has been conducted with the aid of a grant from the Commonwealth Fund for research in dental caries. A report at the last general meeting of the International Association for Dental Research contained a preliminary allusion to a phase of this work: Rosebury, *Journal of Dental Research*, 1930, x, p. 403. Received for publication on October 20, 1930. Presented at a meeting of the New York Section of the International Association for Dental Research on December 10, 1930: *Journal of Dental Research*, 1931, xi, p. 59.

lactobacilli in the mouth (13); (*c*) the inception of the carious process may be encouraged by or require either a rachitic nutritional deficiency (6, 7, 16, 17, 18), or (*d*) a scorbutic nutritional deficiency (26, 11, 12, 8).

Albino rats were selected as the experimental animals because, like human beings, they are omnivorous, and their molar teeth are similar in development and structure to human molars. Their susceptibility to dental caries is indicated by the findings of Grieves (6, 7) and Shibata (24), among others.

TABLE 1

*Group arrangements and designations**

	DIET	SUBGROUPS, MINUS LACTOBACILLI	SUBGROUPS, PLUS LACTOBACILLI
Group I: 5 litters of 4 males each	Control	NR*a*	NR*b*
	Rachitic	R*a*	R*b*
Group II: 5 litters of 4 males each	Control	NS*a*	NS*b*
	Scorbutic	S*a*	S*b*
Group III: 5 litters of 4 females each	Control	NC*a*	NC*b*
	High CHO	C*a*	C*b*

* Each subgroup (cage) included five animals—one from each litter of the group.

II. EXPERIMENTAL PLAN

Three experimental groups, involving sixty rats in all, were arranged as represented in *Table 1*. Each subgroup (cage) included five animals, one from each litter of the group. Each group comprised twenty animals of one sex, including five litters of four rats each. Ten of the animals in Group I were put on a low-calcium diet without vitamin D (R subgroups), and ten on the same diet with the deficiencies supplied (NR subgroups). In Group II, ten animals were put on a diet deficient in vitamin C (S subgroups), and ten kept as controls (NS subgroups). In Group III, ten animals were put on a diet high in carbohydrate, but otherwise complete (C subgroups), and the remaining ten on a normal diet (NC subgroups). In order to include the bacterial factor, i.e., the presence of lactobacilli in the mouth in each of the groups, a further subdivision was made so that half of the

normals and half of the test animals of each group were fed a human oral culture of lactobacilli; while the remaining normals and test animals went without such feeding. Thus each group was divided into four subgroups of five rats, and every subgroup placed into a cage. The litters were disposed so that each subgroup, or cage, contained one member of each litter in the group, to provide direct litter-mate control. In order that part of the period of eruption of the molars would be included in the course of the experiment, the animals were only 18–30 days old, weighing 20–51 grams, when placed on the experimental diets.

The use of rats for experiments involving scorbutic deficiency requires some explanation. The findings of Höjer (9) and others (26, 21), of dental abnormalities in guinea-pigs given dosages of vitamin C previously considered adequate to prevent scurvy, seemed to open the possibility that rats, although insusceptible to the grosser manifestations of scurvy, might nevertheless show scorbutic changes in their teeth. In view of the emphasis recently laid upon the scorbutic factor in caries-susceptibility (11, 12, 8), it seemed advisable to test this point on rats, rather than on guinea-pigs, since the persistently growing molars of the latter animal are not comparable to the human dentition.

III. METHODS

A. Diets and feeding

The diets employed were prepared as far as possible from relatively simple substances, in order to avoid the difficulty of interpreting results obtained with complex "natural" foods. The basal diets were composed as follows (per cent):

Groups	*NR*	*R*	*NS–S–NC*	*C*
Egg albumin	10	10	10	6
Wheat gluten	20	20	20	12
Corn starch	41	41	41	27
Glucose	10	10	10	40
Hydrogenated fat (Crisco)	15	15	13	9
Osborne-Mendel salt mixture	4	—	4	4
Modified salt mixture	—	4.3	—	—
Cod-liver oil	—	—	2	2

Glucose was selected as the sugar because it is readily utilizable by all strains of lactobacilli, while there is considerable variation in the degree to which sucrose is utilized by these forms (23). The Osborne-Mendel salt mixture (20) was selected because of its content of iodine, manganese, fluorine, and aluminum, which might otherwise be lacking from a synthetic diet, and because its calcium is easily removed and replaced, being present as a single salt. The "modified" salt mixture was prepared from the Osborne-Mendel formula, with the following changes. The calcium carbonate was omitted, and the sodium carbonate and potassium carbonate each doubled in amount to restore the acid-base balance. Preliminary trial of the diet including this salt mixture on a group of young rats, with the vitamin additions as described below, indicated that the calcium provided in the spinach and orange juice was not sufficient to sustain life. Accordingly, enough calcium lactate was subsequently added to the diet, before beginning the described experiments, to bring the calcium-phosphorus ratio of the salt mixture to 1:2. Then, to compensate for the greater bulk of this salt mixture as compared with the normal Osborne-Mendel salt, 4.3 parts were used in 100.3 parts of the rachitic diet, instead of 4 per cent. After the animals had been on the R diet for thirteen days, it seemed advisable to improve it further. Accordingly, sodium chloride was added to both R and NR diets to the extent of 1 per cent of the total basal diet, in order to counteract the possibility that an excess of potassium in the rachitic diet was increasing its severity by interfering with calcium absorption (25). The following additional foods were given to supply vitamins:

	Dosage per rat per day	*Subgroups*
Yeast Vitamin-Harris Powder	0.2 gram	All subgroups
Orange juice	2.0 cc.	NR, R, NS, NC, C
Spinach (fresh leaves)	3.0 gram	NR, R
Viosterol (Mead-Johnson 100*D*)	0.002 cc.	NR

The dosage of yeast powder represents approximately four times, and that of viosterol and cod-liver oil eight times, the quantities recom-

mended as the protective minima by their respective manufacturers. The dosage of orange juice was arrived at by conjecture, there being no experimental basis other than the fact (5) that 3 cc. is the minimal fully protective dose for the guinea-pig.

Viosterol (diluted with cottonseed oil) was fed individually by means of a pipette. Cod-liver oil was incorporated in the diets of the groups receiving it, freshly each week. The yeast powder was mixed with the orange juice and the mixture fed separately. In the case of the S group, which did not receive orange juice, the yeast powder was mixed with water. All basal diets and water were fed *ad lib.* The animals were kept in cages provided with wire mesh false bottoms, and weighed individually twice each week.

B. Bacteriological methods

The administration of bacterial cultures entailed the feeding of an additional substance, gum tragacanth, to serve as a medium for the suspension of the organisms, and to secure their retention in the mouth. A paste prepared from gum tragacanth is adhesive in character, slowly soluble in water, and, in the dilution and quantity employed, contains only negligible amounts of utilizable carbohydrate and inorganic salts. A washed culture of human oral lactobacilli, suspended in this paste, had previously been found viable after a period of two months at 37°C., and again after an additional ten months at room temperature. For this work the paste was prepared in bulk, bottled in 40-cc. amounts, and sterilized by the fractional method in an Arnold sterilizer. At intervals of two weeks during the course of the feeding a mass culture of a strain of lactobacilli, isolated from a human case of dental caries by the method described elsewhere (23), was incubated at 37°C. for 24 hours; then centrifuged; washed repeatedly with saline; and the final suspension in a minimum of saline mixed, under aseptic precautions, with the tragacanth paste. The organisms in 200 cc. of broth were divided among five 40-cc. portions of the paste, so that 1 cc. of the mixed paste contained approximately the number of organisms in 1 cc. of broth culture. Each of the *b* cages (*Table 1*) received 15 grams of this paste-suspension, mixed immediately before use, with enough of the basal diet to make a stiff adhesive mass. The control rats were given sterile tragacanth paste

mixed with the basal diet in the same way. In preliminary experiments this paste feeding was attempted every day, but the appearance of diarrhea among the animals made such continuous feeding inadvisable. It was found that the pastes could be fed twice weekly without ill effects, and this procedure was adopted in the described experiments. The basal diets were fed in dry form on the other days of the week.

Before being put on the diets, and at intervals during the course of the feeding, the mouths of all the animals were cultured in the following manner. Small sterile cotton swabs were rotated in the mouth in the region of the molars, and inoculated into 1 percent glucose broth (pH 5.0) containing chopped meat for partial anaerobiosis, and smeared on 1 percent glucose-agar plates (pH 5.0). These were incubated for 48 hours at 37°C. The morphology of the predominantly aciduric flora thus obtained was studied by means of gram-stained smears.

C. Histological methods

At the conclusion of the experiments the surviving animals were killed with chloroform, and their lower jaws and upper molar teeth removed and transferred to 10 per cent formalin. In the first group the tibias were removed from some of the animals, chosen at random; some of these were preserved in acetone for line tests, and others were dried for ashing. The animals that died before the conclusion of the experiment were treated similarly. One lateral half of each mandible was decalcified, embedded in celloidin, and eight 20–30 μ sections cut from it longitudinally, to include the three molars, at different molar levels. The other half of each mandible was embedded in collolith, and similar longitudinal ground-sections prepared, the molars and the coronal half of the incisor having been ground separately. All sections were stained with hematoxylin and eosin.

IV. RESULTS

A. Growth

The accompanying graphs illustrate the growth in the different subgroups (*fig. 1*). The curve in each case represents the average of the subgroup, and the points above and below it represent extreme weights

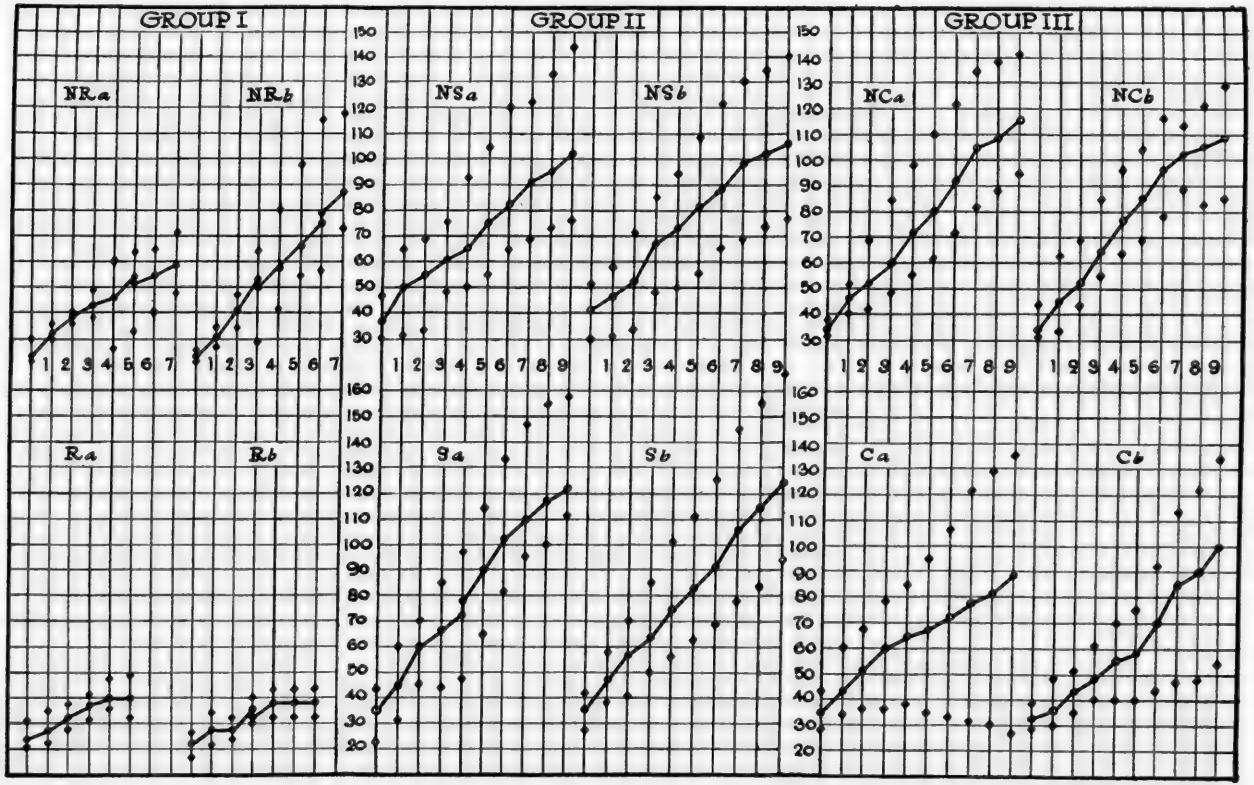

FIG. 1. Mean growth curves of all subgroups. The points above and below each curve represent the extremes of the subgroup. Abscissas = weeks; ordinates = grams; N = controls; R = rachitic group; S = scorbutic group; C = high-carbohydrate group; *a* = diets fed without lactobacilli; *b* = diets fed with lactobacilli. Example: NS*b* = controls of scorbutic group, with lactobacilli. (See table, p. 122.)

in the group. The animals in Group I were started at lower weights than those of the other groups. Since it was generally true that the animals of smallest initial weight showed the poorest development during the experimental period in all groups, this circumstance may have retarded development in the NR subgroups, and aggravated the severity of the diet in the R subgroups. The graphs indicate general slight superiority of growth in the *b* subgroups, particularly NR*b*, over the respective *a* subgroups. This may be fortuitous, or it may be due to some nutritive factor in the bacterial cells.

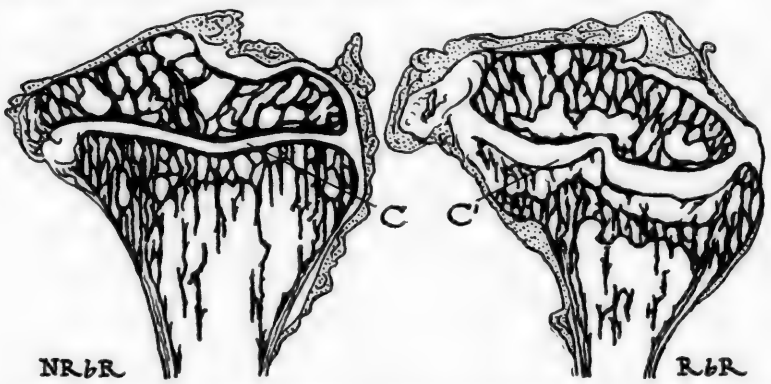

FIG. 2. Line tests. Upper extremities of tibias on control and rachitic diets, split and impregnated with silver. (Line tests.) Note that the epiphysial cartilage of the latter (C′) is broader and less regular than that of the control (C).

The animals in the R subgroups, although they presented scrawny fur and an undernourished appearance, did not manifest the lameness characteristic of rickets. Their tibias, however, when treated with silver nitrate, presented a picture sufficiently different from that of the normal rats to indicate deficient calcification. Whereas, in the normals, the cartilaginous junction of head and shaft is narrow and regular, in the test animals it appears wider and distinctly irregular (*fig. 2*). This line test is known to be given much less clearly in the low-calcium type of rickets, as in this instance, than in the low-phosphorus type, in which it presents a marked variation from the normal. That the animals on the R diet developed rickets is further attested

by the following determinations, which represent percentages of ash from the dried alcohol-ether-extracted tibias.

Controls	*Percent*	*Test animals*	*Percent*
NR*a*L	47.3	R*a*L	27.2
NR*a*R	52.3	R*b*L	33.4
NR*b*L	53.0	R*b*O	29.5
		R*b*RL	27.0

In the second and third groups, growth in general was good. In Group II, where no difference in growth was to be expected between the normal and the test animals, the curves indicate a slight superiority in the subgroups on the scorbutic diet. In Group III the controls averaged slightly better than the test animals. The difference here appears to be due to the erratic growth of three animals in the C*a* and C*b* groups, one of which, whose growth is represented by the lower points in the C*a* graph, weighed less at the conclusion than at the beginning of the experiment. In this instance, again, it was the smallest animals that were affected. The lower percentage of protein in the *C* diet, and its correspondingly lower nutritive ratio, may have influenced this result.

The surviving animals in the NR subgroups were killed after being on the diet 46 days; those in the R subgroups, after 40. The animals in the second and third groups were killed after 60 days.

B. Bacteriological findings

According to the plan of these experiments, the feeding of lactobacilli in suspension in tragacanth paste was to have been an attempt at the oral implantation of these organisms. It was recognized that the feeding of broth or milk cultures, both of which have been attempted (10. 1, 4) with the same purpose, was open to the objection that such media may pass rapidly through the mouth and there affect the resident flora little or not at all. We hoped that the combination of an adhesive menstruum, which would remain in the mouth somewhat longer at least than a liquid, with diets affording readily utilizable carbohydrate, might solve this problem. Two findings, however, made this hypothesis, on the one hand doubtful, and on the other apparently superfluous. In the first place, observation disclosed that

the pastes as fed, incorporated with the dry diets, were not being retained in the mouths of the animals in visible amounts. It can hardly be doubted that the natural cleansing mechanism of the rat mouth is greatly superior to that of man. This matter is discussed more fully below.

In the second place, the observation was made on examining the first group of rat-mouth cultures, and confirmed on all such examinations subsequently, that aciduric gram-positive rods, indistinguishable morphologically from *L. acidophilus*, are a normal inhabitant of the rat mouth.[2] Several isolations of these organisms were made, but no detailed study has yet been undertaken to determine their relation to human oral lactobacilli, beyond the following facts, which, so far as they go, make it unlikely that we are dealing with an unrelated form: In general appearance, including size, grouping, and morphological variability, and in their colonies in glucose-agar pour plates, which are minute and show both the rough "X" and the smooth "Y" forms, they are characteristically lactobacilli. In addition, in cultures from the rat mouth into pH 5.0 partially anaerobic broth, they appear invariably as the predominating form; after a 24-hour incubation at 37° C. they usually occur alone, other forms appearing after an additional 24 hours. A single series of fermentation tests with seven isolated strains of these organisms yielded the following results:

Strain	Glucose	Maltose	Lactose	Sucrose
R*b*RL	+	+	+	−
NS*a*LL	+	+	+	+
NS*b*L	+	−	−	−
S*a*L	+	+	+	+
S*a*RL	+	+	−	+
S*b*R	+	+	+	+
S*b*LL	+	+	+	+

To check the possibility that the uniform occurrence of organisms of this type, in the mouths of the animals used in this work, might depend on some peculiarity associated with the stock (all of these animals having been obtained from one source), a group of fourteen rats and one guinea-pig belonging to Dr. W. H. Eddy of Teachers College, Columbia University, bred by him through several genera-

[2] See Klein and Harris: *Journal of Dental Research*, 1931, xi, p. 149. (This issue).—[*Ed.*]

tions and living on a variety of experimental diets, were cultured in the same way. All but one rat yielded the same gram-positive rods, but since the method of culturing used is not infallible, the exception is hardly significant. As a matter of conjecture it appears reasonable that *L. acidophilus* should appear in the rat mouth. The species is widespread in the intestines of animals, and the rat mouth is exposed to continuous fecal contamination in spite of the presence in the cages,

FIG. 3. Retention of debris in molar pits. *SaR*—Scorbutic diet. Upper-molar pits filled with amorphous debris.

as in these experiments, of wire-mesh false bottoms. Therefore, the intestinal influence on the rat-mouth flora is probably profound, constituting an additional difference from the human mouth. The fact that gram-negative rods, of the *B. coli* type among others, were also prominent in the rat-mouth cultures in this work, supports the view.

Since it was impossible to distinguish morphologically between the lactobacilli native to the rat mouth and the human strain which was fed, no check on the success or failure of the attempted implantation

could be made. On the other hand, as has been stated, the attempt now seemed superfluous; but since it has not been established that important differences do *not* exist between the rat organisms and those associated with dental caries, the feeding of human oral organisms was continued throughout the experiment.

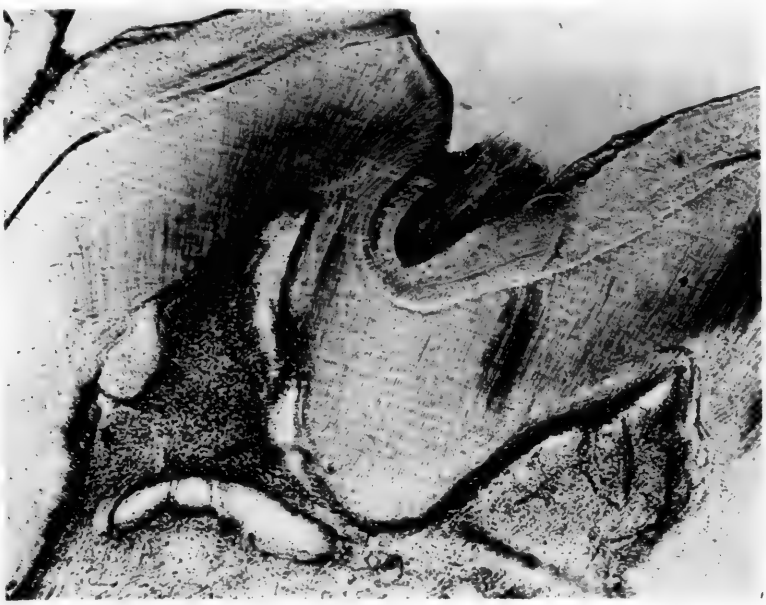

FIG. 4. Retention of debris in molar pits. C*bR*—High-carbohydrate diet. Lower-molar pit filled with fibrous debris.

C. Histological findings

Completely negative results were obtained in this work insofar as the attempted production of dental caries is concerned. On examination of the molars under a dissecting microscope before preparing the jaws for sectioning, most of the molar pits presented an aspect of discoloration generally similar to incipient caries in human molars, but the sections revealed that these were pits filled with debris, the enamel and dentin at their bases being normal (*figs. 3* and *4*). In many cases the amorphous character of the material that filled the pits suggested

Nasmyth's membrane, which in these cases might be considered protective; but in others distinctly fibrous tufts, masses of vegetable cells, and even scraps of an opaque material, which is probably metal gnawed from the cages, were found. It is likely, therefore, that food impaction in these pits is not prevented, in all cases at any rate, by a natural protective agency. This possibility is discussed below. Other histological findings, dealing with the effects of the diets employed on the structure of the teeth, are presented separately in the succeeding paper.[3]

V. DISCUSSION

In consideration of the results obtained in this work from the viewpoint of susceptibility to dental caries, certain interesting implications arise. In all of the animals it is apparent that a combination of two of the important factors in the mechanism of caries according to Miller (19)—the presence in the mouths of fermentable carbohydrate and of actively acidogenic bacteria—with, in addition, the presence of retentive molar pits, did not give rise to caries. And, in the three groups respectively, it is also interesting that poor calcification of the teeth in one case, one variety of pulpal impairment in another,[2] and in the third the presence of a high-glucose diet, which might be expected to provide a distinctly favorable environment for an acidogenic flora, each added to the preceding factors, still did not induce the carious process. It is, of course, hazardous to draw conclusions from such negative findings. Yet, in so far as they apply to dental caries in the rat, we believe these findings indicate that the factors herein applied to the problem do not in themselves provide the complete combination of causes of caries susceptibility.

It has been stated that the attempt made in this work to foster the retention of food and acidogenic bacteria about the teeth does not appear to have been successful. Considering the apparently high efficiency of the normal cleansing mechanism of the rat mouth, it is conceivable that this is the crux of the reported failure; although it is not easy to reconcile this with the evidence of food impaction presented. Further work along these lines is being planned.

[3] Rosebury and Karshan: *Journal of Dental Research*, 1931, xi, p. 137.

VI. SUMMARY

Sixty young rats were studied in three groups, in which were tested, respectively, a low-calcium, vitamin D-free diet, a vitamin C-free diet, and a diet high in fermentable carbohydrate (all synthetic), in each case with and without the addition of human oral lactobacilli fed in an adhesive paste. All rat mouths were cultured at intervals for aciduric organisms.

The character of the growth obtained with the different diets is recorded. The presence of rickets in the first group was ascertained by line tests and ash determinations on the tibias.

Aciduric bacteria similar to *Lactobacillus acidophilus* were found to be a normal inhabitant of the rat mouth, their presence being constant and independent of the feeding of human lactobacilli.

No dental caries was produced, in spite of evidence of retention of food in molar pits, probably indicating the absence in these experiments of one or more factors in susceptibility to caries.

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PATHOLOGICAL CHANGES IN THE TEETH OF RATS PRODUCED BY SYNTHETIC DIETS[1]

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I. INTRODUCTION

In the preceding paper[2] we described experiments that included the use of synthetic diets in an attempt to produce dental caries in young albino rats. The present paper deals with the histo-pathological changes produced in the teeth and jaws by these diets. In recording these findings, significance has been attached only to conditions that were uniformly present in all of the animals reared under a given set of experimental conditions. Abnormalities occurring in isolated instances have been omitted. In most cases, these occasional findings appertain to animals that died before the conclusion of the experiment, and are to be considered either as post-mortem changes, or as conditions arising previous to death and owing to the same cause.

II. CHANGES ON THE LOW-CALCIUM, VITAMIN D-FREE DIET

The animals reared on the low-calcium, vitamin D-free diet showed certain well-defined abnormalities of molars, incisors, and bone, which are illustrated in *figs. 1–6*. This condition was present in all of the

[1] This work has been conducted with the aid of a grant from the Commonwealth Fund for research in dental caries. Received for publication on October 20, 1930. Presented at a meeting of the New York Section of the International Association for Dental Research on December 10, 1930: *Journal of Dental Research*, 1931, xi, p. 60.

[2] Rosebury and Karshan: *Journal of Dental Research*, 1931, xi, p. 121.

animals fed this deficient diet, including those that survived the duration of the experiment, and those that died. The group controls were entirely free from it.

In the incisors, the pulps, including the odontoblasts, appear normal. The odontogenic zone of the dentin is abnormally broad, occupying about half the width of the dentin, and is separated from a narrow peripheral band of apparently normal dentin by a zone of large calcified globules. Dentinal tubules may be traced through the tissue. At different levels the layer of globules varies in breadth; at the apical end of the incisor it is narrow, while anteriorly it assumes great prominence. The condition occurs in general throughout the length of the incisor. The incisal enamel matrix, as it appears in decalcified sections toward the apical end of the tooth, shows considerable irregularity of contour. Apically it stains too deeply to show structure; but, near the point where it thins out to be replaced by fully calcified enamel, its structure appears disorderly, the prisms being disposed in soft, broken curves, or too irregularly to be traced. The ameloblasts are irregular because of their alignment along the deranged outer enamel edge, but they show in addition what seem to be intrinsic irregularities—shrinkage and distortion of cells—varying in degree at different levels. Here and there a brief column of regular ameloblasts occurs.

In the molars, there is likewise no pulpal deformity. The dentin appears similar to that of the incisors, except that only occasional globules occur. The odontogenic zone is again notably broad throughout its extent. The peripheral dentin is finely mottled by the occurrence of interglobular spaces, but these occur to an apparently equal extent in many of the normal controls of this group. Enamel keratin is present in abundance, indicating that its proportion is probably higher in the rat than in man (11); but no information can be gained from it regarding the condition of the enamel. In the ground sections, as will be noted, the molar enamel in the rachitic animals is indistinguishable from the normal.

The cancellated alveolar bone shows a characteristic change in the rachitic animals. The trabeculae, although normal in cellular structure, present central areas stained with hematoxylin, their peripheries being eosinophilic. The compact bone under (external to) the incisor is stained similarly. This is in contrast to the bone of the control

animals, in which traces of hematoxylin staining occur, but always as narrow lines at the periphery of the trabeculae, the matrix being evenly eosinophilic.

In the ground sections, the general picture of this deformity differs in that calcified areas of tooth and bone stain poorly or not at all; but the condition remains the same. The broad odontogenic zone in

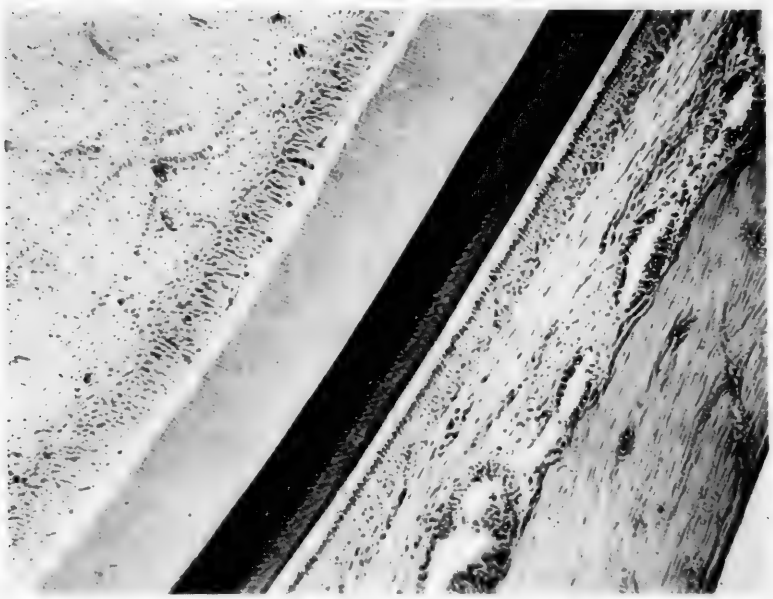

FIG. 1. NRδR—Control diet: low-calcium, vitamin D-free group. Lower edge of incisor, apical region, to show normal structures. Odontoblasts are cut somewhat obliquely. (Low power.)

both molars and incisors is deeply stained, and the bone stains peripherally (with eosin) rather than centrally. No abnormality is apparent in the fully formed coronal enamel of the incisor, but irregularity begins to appear beneath the gingival margin, and is clearly in evidence apically. The molar enamel, however, seems normal. Changes similar to these, in the teeth of rats, have been described by Bracco (1), Orban (8), and, very recently, Weinmann (12). All these workers studied only the incisor teeth. Bracco and Orban used

natural-food diets; details of the diets used by Weinmann are not given. The results obtained in this work are therefore, we believe, more directly attributable to a specific dietary deficiency than those of the previous workers, inasmuch as synthetic diets were used by us. The changes in the molars have apparently not been described hereto-

FIG. 2. NR*b*O—Low-calcium, vitamin D-free diet. Lower edge of incisor, apical region, to show changes described. Odontoblasts are cut obliquely. (Low power.)

fore. The results obtained by Mellanby, in rickets in dogs (7), are generally the same as those described above.

III. CHANGES IN THE OTHER GROUPS, INCLUDING THE VITAMIN C-FREE DIET

In the second and third groups, which comprised subgroups on the vitamin C-free diet, the high-carbohydrate diet, and their respective controls, a deformity was present in the molar teeth of all forty ani-

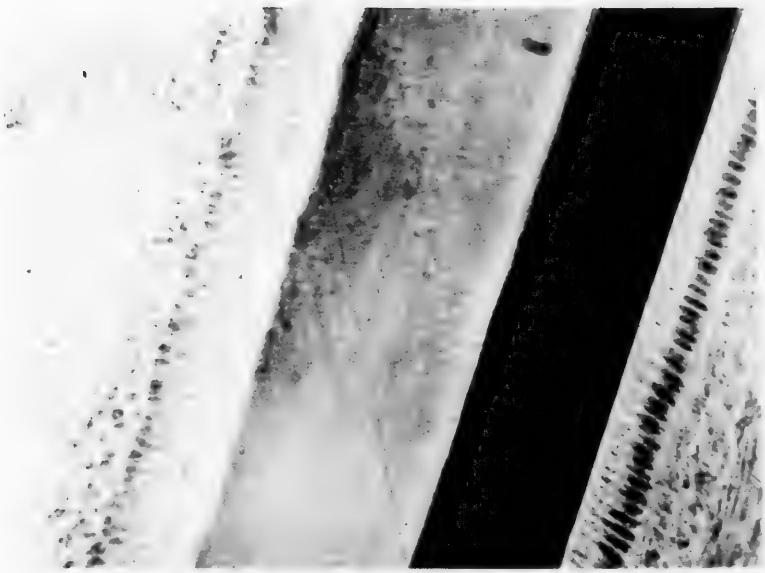

FIG. 3. NR*b*RL—Control diet: low-calcium, vitamin D-free group. Lower edge of incisor, apical region, to show normal structures. (High power.)

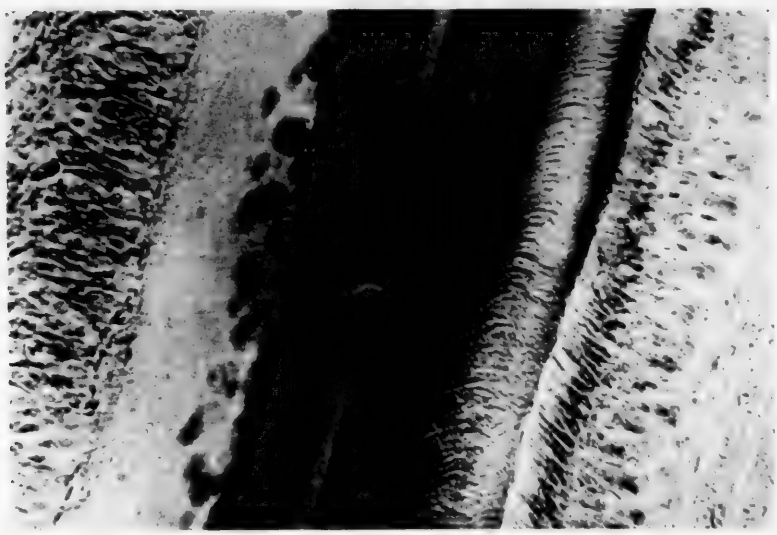

FIG. 4. R*a*R—Low-calcium, vitamin D-free diet. Lower edge of incisor, apical region, to show changes described. Odontoblasts are cut obliquely. (High power.)

mals (*figs. 7–9*).[3] The changes occurred only in the molars, the incisor teeth of these animals having been entirely normal. No significant differences in kind or degree of involvement appeared in any of the individual subgroups. All of the molars were affected to some degree, but in many cases one of the three was more markedly involved than the others. In its fully developed form, the condition is generalized over the whole pulp, although osteoid and nodule formation oc-

FIG. 5. NR*b*L—Control diet: low-calcium, vitamin D-free group. Molar root and alveolar bone, to show normal structures.

curs chiefly in the pulp chamber proper; and, while in some cases the deformity extends in extreme form to the apical foramen, in others the apical portion of the root may be quite normal in teeth otherwise badly affected.

The dentin, in the half of its breadth adjoining the pulp, presents a series of unevenly stained irregular laminations, which seem to be

[3] Figures 3 and 4, in the preceding paper in this issue (footnote 2), show these changes as they appear in ground sections.

generally independent of tubular deflection. The pulpal edge of the dentin is thrown into uneven contours following these laminations. In one or more places in the coronal pulp, in many of the teeth, localized masses of secondary dentin appeared as excresences on the pulpal wall, continuous with the main body of the dentin. In general these have a more or less regular tubular (dentinoid) structure. In

FIG. 6. R*b*O—Low-calcium, vitamin D-free diet. Molar root and alevolar bone, to show changes described. Section cut obliquely.

addition, in most of the teeth, there were present masses of tissue of an osteoid character, of varying size, generally free in the pulp chamber, but sometimes in contact with the wall. Where such contact occurs, however, the osteoid mass may be sharply differentiated from the underlying dentin, being separated by a layer of apparently crushed cells. Although bone cells in lacunae occur in these masses, no lamellae or haversian canals are present. Here and there a small island of pulp cells appears, enclosed within the osteoid tissue. The odontoblasts appear to be normal in those places where the pulpal wall preserves a fair degree of regularity. In a few regions of pronounced

FIG. 7. NSALL. Control diet: vitamin C-f[illegible] Low power view of molar, to show changes described. Section cut somev[illegible]

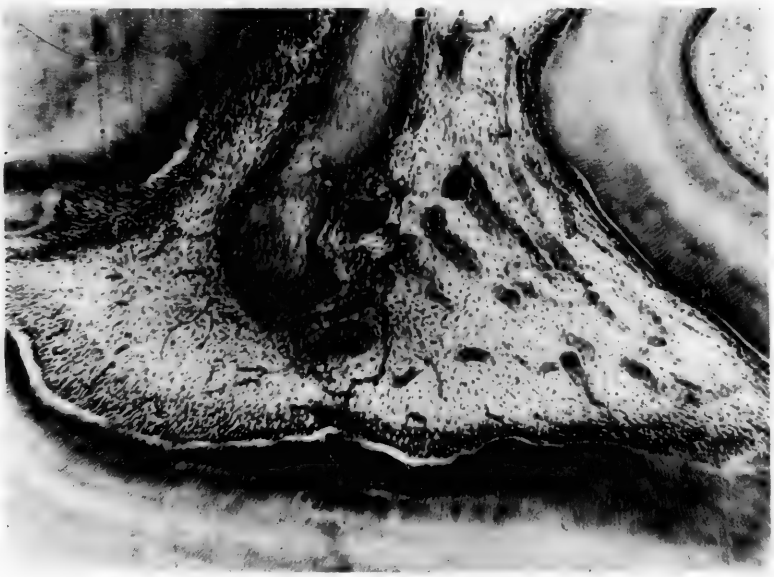

FIG. 8. NSALL.—A portion of the field shown in *fig. 7*, further magnified.

irregularity, they seem to be altogether lacking. In general, where the pulpal wall is irregular, the odontoblasts are likewise irregular in arrangement; but, allowing for the difficulty of interpreting sections of cylindrical cells, whose form seems to change with the plane of section, they appear healthy and not abnormal. The cellular structure of the pulp seems in general to be normal; but, in the neighborhood

FIG. 9. S8R—Vitamin C-free diet. Lower edge of incisor, apical region. Normal structures.

of osteoid masses, the pulp tissue appears denser than elsewhere, so that an appearance of compression is produced, and the cells in the immediate vicinity of the masses are difficult to identify. Some of these, however, have the appearance of osteoblasts.

IV. DISCUSSION

The disorders occurring in the group on the low-calcium, vitamin D-free diet, excepting possibly those of incisal enamel and ameloblasts, are all explainable as owing to deficient calcification. The character

of the enamel involvement suggests two additional possibilities of histogenetic interest. In the first place, since the dentin involvement is generalized throughout that tissue, while only the youngest enamel (which might have been formed during the period of experimental feeding) is affected, it appears that dentin, although previously formed and calcified, may be capable of undergoing a process of decalcification during life, under the influence of a deficient diet; whereas fully formed enamel may be impervious to such influence. The fact that the dentin, though complete, retains association with the living tissue (the pulp) which forms it, while the enamel, when fully formed, discards its formative organ, bears out such a view. In the second place, if the fundamental alterations produced in these teeth are conditioned by imperfect calcification, a comparison of deformed ameloblasts with apparently normal odontoblasts suggests that the process of calcification in enamel is intimately associated with ameloblast function, whereas the analogous process in dentin is independent of the function of the odontoblasts. In this connection it is noteworthy that the recorded abnormality of incisal enamel is morphological, whereas that of the dentin does not seem to have affected the form of the tissue. Hopewell-Smith (5) and Orban (9), and others, have expressed views similar to this, based on evidence of a different nature.

With respect to the disorders occurring in the teeth of the second and third groups, their relationship to the picture of latent scurvy in the teeth of guinea-pigs as described by Zilva and Wells (13), Robb, *et al.*, (10), and especially by Höjer (4), merits consideration. While these experiments were in progress, Eddy (3) stated that rats may be susceptible to scurvy of this sort; and somewhat more recently, Kotanyi (6) reported a similar finding. The results obtained in this work, nevertheless, do not appear, on analysis, to bear out this view. The deformity described here is similar in some respects to that described by Höjer (4), but the two are not identical. Höjer's picture seems to be fundamentally that of an odontoblastic metamorphosis into cells resembling osteoblasts, and producing a tissue, continuous with the formed dentin, resembling bone. The deformity described here, on the other hand, seems to involve the odontoblasts only secondarily, and the adventitious product is merely a somewhat disordered dentin; but osteoblast-like cells appear *within* the pulp and there produce

masses of an osteoid tissue, which do not achieve continuity with the formed dentin. These differences, nevertheless, may be matters of degree, or of species dissimilarity between the rat and the guinea-pig. But in Höjer's instance the incisors are chiefly involved, and the absence of incisal abnormality is hardly explainable on similar grounds.

It seems still more significant that the described condition occurred not only among animals reared upon a completely scorbutic diet, but to an equal extent in the controls, and also in the entire third (high-carbohydrate) group—animals that received a supposedly adequate ration of vitamin C. It will be noted that the diets[4] given to three of the four sub-groups involved—the NS, S, and NC subgroups—were identical except for the omission of orange juice in the case of the S subgroup; and that the diet of the C subgroup did not differ in the matter of accessory foods. The diets fed to the NR and R subgroups, on the other hand, were different from the others in some respects, particularly in that the former received spinach. It is interesting to observe that, in the NR and R subgroups, the secondary formations are totally absent in most instances, although present in very mild form (dentinal irregularity only, without nodule or osteoid formation) in seven cases, and in more pronounced form in two.

The explanation of these findings must await further study. At present the possibility that they represent scorbutic changes seems remote, although it cannot be altogether excluded. It seems more likely that we are dealing with a deficiency which was not supplied by orange juice, but which may have been supplied by the spinach fed to the first group, to an extent just short of full protection. The recent work of Dalldorf and Zall (2) in which mixed green vegetables, including spinach, were found to be more effective than orange juice in preventing the dental manifestations of guinea-pig scurvy, may be considered as supporting this view.

V. SUMMARY

A synthetic diet low in calcium and free from vitamin D was found to produce changes in the teeth of rats, ascribable to deficient calcification, as follows: The odontogenic zone is abnormally broad in the dentin of both molars and incisors, and large calcified globules appear

[4] See the preceding paper in this issue (footnote 2).

in the body of the dentin. The enamel of the molars, and the fully formed enamel of the incisors, are apparently normal; but the youngest enamel of the incisors, and the apical ameloblasts, present evidence of deformity. An alteration in the staining quality of the alveolar bone occurs. The pulps, including the odontoblasts, remain normal. Histogenetic implications of these findings are mentioned.

Other synthetic diets, including a vitamin C-free diet, and diets in which vitamin C was supplied by 2 cc. of orange juice per rat per day, were found to give rise to changes in the pulps and dentin of the molars only, similar in some respects to the manifestations of latent scurvy in the teeth of guinea-pigs. These changes do not seem to be due to vitamin-C deficiency; their specific cause has not yet been determined.

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A NOTE ON THE PRESENCE OF ACIDURIC BACTERIA IN THE MOUTHS OF RATS

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In executing a series of diet experiments on rats in the McCollum colony it has been found that aciduric organisms are present in the

FIG. 1. Small gram-positive rods from 24-hour tomato-broth culture of swab from rat mouth, showing palisade arrangement of the bacilli. ×1800

mouths of stock rats.[1] These organisms grow well at pH 4.5 and resemble closely the aciduric organisms of the *Lactobacillus acidophilus* type. Two different morphological types are found. One is

[1] Rosebury has recently reported similar findings: *Journal of Dental Research*, 1930, x, p. 404. [See also this issue: *Ibid.*, 1931, xi, pp. 130–131.—*Ed.*]

a short rod with a characteristic palisade arrangement (*fig. 1*), almost identical with those described by Bunting from human mouths, while the other form is a much longer rod often found in long chains and

FIG. 2. Large gram-positive rods from 24-hour tomato-broth culture of swab from rat mouth, showing chain formation and curved forms. ×1800

occasionally in filaments (*fig. 2*). Curved forms and rods with swollen ends are also seen in smear preparations of the latter type. Both types are gram-positive.

THE HISTOPATHOLOGY OF EXPERIMENTAL MOLAR CARIES IN RATS[1]

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An extensive array of direct experimental evidence shows that defective tooth-structure results from the feeding of deficient diets. However, few workers have observed actual cavity production as a result of such feedings. McCollum and co-workers (3) reported the finding of macroscopic cavities (caries-like lesions) in the molars of rats on diets unbalanced in protein and mineral contents. Marshall (5) and Bunting (2) found cavities histologically in rats on diets low in calcium and vitamin A. Howe (4) observed similar lesions in guinea pigs deprived of this vitamin. Bloch (1), however, was unable to find dental caries in cases of xerophthalmia caused by deficiency in vitamin A.

In the course of an investigation on the effects of unbalanced diets on the teeth of rats, it became apparent that frequent caries-like lesions were present in the molars of rachitic rats which had been fed viosterol therapeutically for long periods. The present communication is concerned with a description of the histology of such cavities.

The rats used were the offspring of healthy females, which had been receiving diets adequate in all respects. At weaning the young were placed on a rachitogenic diet, and continued on this diet for nineteen days. At the end of this time 1 per cent of viosterol, equivalent to cod-liver oil,[2] was added to the ration, which was fed for 87 days, until the rats were 135 days old. When the teeth of these rats were examined, the most striking defect noted was an extensive

[1] Aided by a grant from the Mead Johnson Company, Evansville, Indiana, and the Research Commission of the American Dental Association.

[2] The viosterol was diluted so that 1 cc. equalled 1 cc. of cod-liver oil therapeutically.

FIG. 1. EXPERIMENTAL CARIES IN RATS ON RACHITOGENIC DIET AND VIOSTEROL

A—food débris in cavity of second molar. *B*—attempt to wall-off cavity. *C*—beginning caries in right cusp of second molar. Areas 2, 3, 4, and 5 are shown in *Figs. 2, 3, 4,* and *5* at higher magnifications.

FIG. 2. MAGNIFICATION OF AREA 2 IN FIG. 1

A—disappearance of odontoblasts. *B*—degeneration and replacement by areolar connective tissue. *C*—attempt to wall-off cavity. *D*—streaming of fibrous tissue. *E*—piece of broken-off dentine.

destruction of the coronal surfaces of the mandibular molars. The mandibles sectioned and examined histologically gave a picture that simulates caries found in human beings.

FIG. 3. MAGNIFICATION OF AREA 3 IN FIG. 1

A—food débris in cavity. *B*—tightening of connective-tissue band. *C*—proliferation of fibrous tissue. *D*—disappearance and calcification of odontoblasts.

Fig. 1 illustrates the condition found in the second and third mandibular molars of the experimental animals. The second is extensively

FIG. 4. MAGNIFICATION OF AREA 4 IN FIG. 1

A—early caries in crown of molar. *B*—fibrous connective-tissue proliferation. *C*—lymphocytic infiltration. *D*—beginning degeneration of odontoblasts. *E*—streaming of finely-meshed fibrous tissue.

involved in the carious process, while the third shows beginning caries at *C*. In the second molar at *A*, the cavity is filled with food débris,

and at *B* a reaction to wall-off the cavity with a connective-tissue band may be observed. Higher magnifications of areas 2, 3, 4,

FIG. 5.—MAGNIFICATION OF AREA 5 IN FIG. 1

A—disappearance of odontoblasts. *B*—lymphocytic infiltration. *C*—dissolution of odontoblasts. *D*—capillary invasion toward affected side of pulp.

and 5, which are given in *figs. 2, 3, 4,* and *5,* demonstrate the tissue reactions in the pulp of the affected teeth.

Fig. 2, representing a magnification of area 2 in *fig. 1,* shows the

attempt of the pulp to form a protective barrier *C* against further invasion of the tooth. At *A* a total disappearance of the odontoblasts has occurred, and at *B* there is noted what appears to be a degeneration of the pulp into areolar connective tissue. Streaming of thickened, fibrous, connective tissue toward the cavity opening is noted at *D*.

Fig. 3, a higher magnification of area 3 in *fig. 1*, shows at *B* an attempt to wall-off the cavity; at *C* a proliferation of connective tissue; and at *D* the disappearance of the odontoblasts and their replacement by amorphous calcified material.

The third molar in *fig. 1* shows beginning caries under the mesial cusp. The caries has not begun in the fissure, but on the higher portion of the crown. *Fig. 4*, an enlargement of area 4 in *fig. 1*, shows the tissue reactions in the pulp. One sees fibrous connective-tissue proliferation at *B*; large numbers of lymphocytes at *C*; beginning degeneration of the odontoblasts at *D* and a streaming of finely-meshed fibrous tissue toward the cavity opening at *E* in an attempt to wall-off the defect.

Interesting tissue reactions are also noted in the pulp distally, under the unaffected portion of the third-molar crown (*fig. 5*). Here an invasion of a great number of blood vessels *D* toward the affected horn of the pulp can be seen. A large number of lymphocytes are noted at *B*, dissolution of the odontoblasts at *C*, and their disappearance at *A*.

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HYPERCEMENTOSIS IN RELATION TO UNERUPTED AND MALPOSED TEETH

A Preliminary Report

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I. INTRODUCTION

Excessive growth of cementum, which can be readily observed through either an unaided ocular examination of extracted teeth (*figs. 1* and *2*), or a roentgenographic examination (*figs. 3–10*), is often referred to as hypercementosis. This condition has been designated by various other terms, among which dental exostosis, execementosis, hypertrophy of cementum, cementum hyperplasia, cementosis, and exostosis are the most familiar. A slight increase in the number of lamellae or layers of cementum not easily detected by visual inspection, but discoverable only by microscopic study, is not considered in the present work as hypercementosis. Cement hyperplasia or hypercementosis has been observed on both vital teeth (*figs. 3–5*) and non-vital teeth, i.e., devital, pulpless, or so-called "dead" teeth (*figs. 6, 8–10*), whether they presented pathological signs (*figs. 5, 7–10*) or not (*fig. 6*).

The subject of hypercementosis has received considerable attention, having been studied by a number of investigators; the final word, however, has not been said. That this is so becomes apparent from the fact, for instance, that there is no unanimity of opinion as to

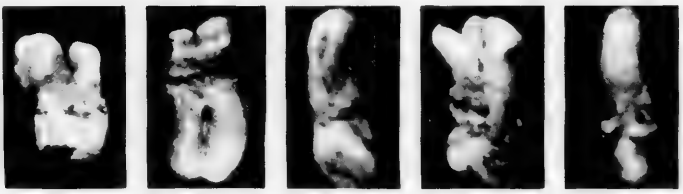

FIG. 1. PHOTOGRAPHS OF EXTRACTED MAXILLARY AND MANDIBULAR TEETH, SHOWING VARIOUS DEGREES OF HYPERCEMENTOSIS

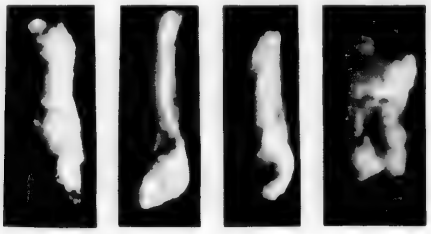

FIG. 2. PHOTOGRAPHS OF UNERUPTED, IMPACTED, AND MALPOSED TEETH REMOVED FROM MANDIBLE AND MAXILLAE

These teeth show presence of hypercementosis

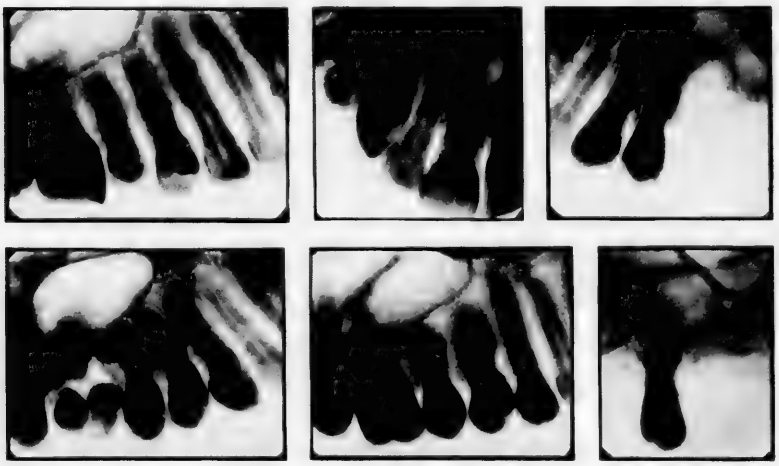

FIG. 3. ROENTGENOGRAMS OF NORMALLY ERUPTED, *vital, healthy* ANTERIOR AND POSTERIOR MAXILLARY TEETH, PRESENTING MARKED HYPERCEMENTOSIS

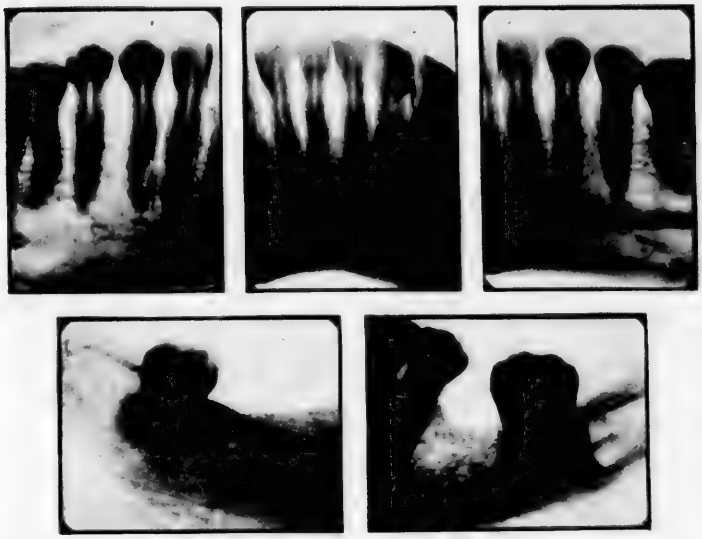

FIG. 4. ROENTGENOGRAMS OF NORMALLY ERUPTED *vital, healthy* ANTERIOR AND POSTERIOR MANDIBULAR TEETH, PRESENTING HYPERCEMENTOSIS IN VARYING DEGREES

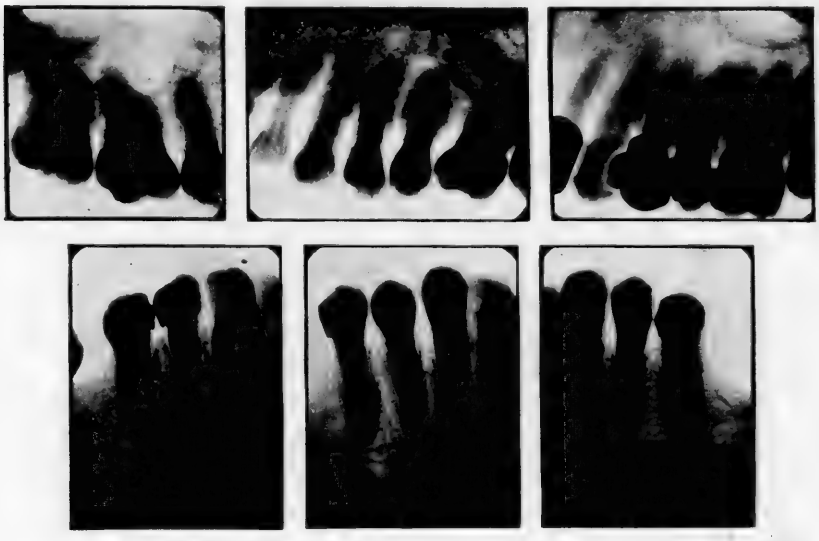

FIG. 5. ROENTGENOGRAMS OF NORMALLY ERUPTED *vital, filled* MAXILLARY AND MANDIBULAR ANTERIOR AND POSTERIOR TEETH, PRESENTING MARKED HYPERCEMENTOSIS
These teeth show no signs of any periapical involvement

what causes hypercementosis. Some ascribe it to purely local factors, such as mild forms of chronic periapical and parietal infection, or low-grade irritation produced by various types of trauma, such as undue

FIG. 6. ROENTGENOGRAMS OF MAXILLARY AND MANDIBULAR TEETH, SHOWING MARKED HYPERCEMENTOSIS

Owing to extensive *caries*, teeth were crowned. No evidence of periapical involvement

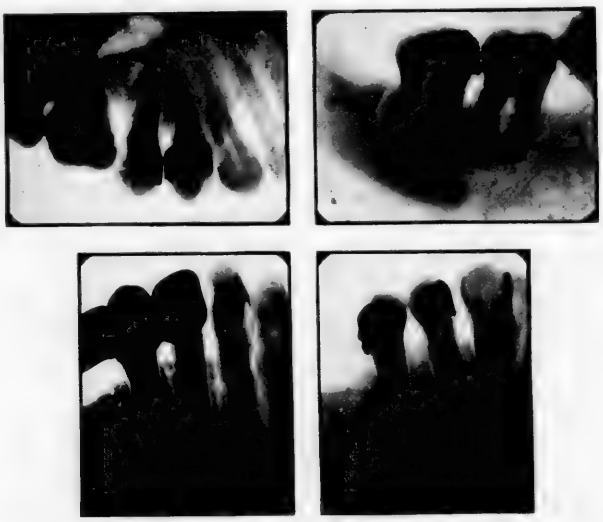

FIG. 7. ROENTGENOGRAMS OF *non-vital, filled* MAXILLARY AND MANDIBULAR TEETH, SHOWING HYPERCEMENTOSIS

These teeth present areas of periapical rarefaction, indicating periapical disturbance

occlusal stress; in this connection excessive function or functional stimulus is held to be an important causative factor. One group of writers attribute hypercementosis to a systemic condition. The

literature reveals disagreement not only as to the causes of hypercementosis but also as to its nature, i.e., whether pathological or physiological, both views having ardent supporters.

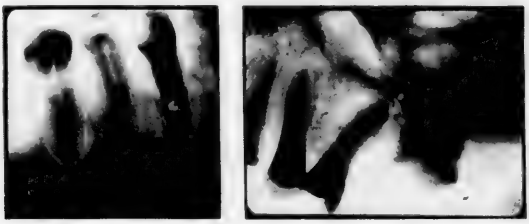

FIG. 8. ROENTGENOGRAMS OF *non-vital, carious* MAXILLARY AND MANDIBULAR TEETH, SHOWING HYPERCEMENTOSIS

These teeth present areas of periapical rarefaction, indicating involvement of surrounding bone.

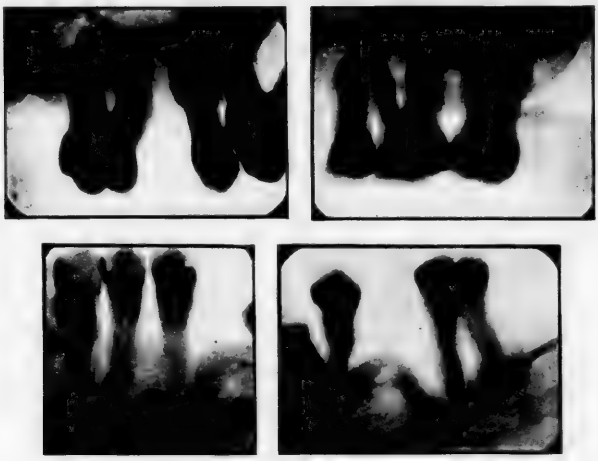

FIG. 9. ROENTGENOGRAMS OF *vital* MAXILLARY AND MANDIBULAR HYPERCEMENTOSED TEETH, SHOWING EXTENSIVE DESTRUCTION OF THE ALVEOLAR BONE (PYORRHEA)

Among the investigators who consider hypercementosis to be pathological, Hopewell-Smith (7) occupies an important position, his definition of hypercementosis as "a *pathological* overgrowth of cementum"

clearly indicating his point of view.[1] Pollia, in his recent book on alveolo-dental radiology (10) refers to hypercementosis as "*pathological* cementum."[1] Bunting (3) also considers hypercementosis pathological having written: "It is often difficult to determine at just what point the formation of cementum ceases to be normal and becomes *pathologic*."[1] He refers to an overgrowth of cementum, i.e., hypercementosis. The fact that hypercementosis is regarded by many as pathological has also been noted by Orban (9). In his book he points out that "a great number of investigators and clinicians believe that cementum hyperplasia is pathologic." He personally, however, is of a different opinion, considering hypercementosis a healthy and desirable condition, thus being in full agreement with Gottlieb (6).

FIG. 10. HYPERCEMENTOSED *non-vital* MAXILLARY AND MANDIBULAR TEETH, SHOWING EXTENSIVE DESTRUCTION OF THE SURROUNDING ALVEOLAR BONE

There are, then, two diametrically opposite views regarding the nature of hypercementosis. In one opinion, prevalent in America, hypercementosis is pathological and requires surgical elimination; in the other, held by the Vienna school, hypercementosis is not only non-pathological but is a favorable condition, and consequently calls for no surgical interference of any kind. An illustration indicating its desirability was given in Gottlieb's statement, before the Pathodontia Section of the First District Dental Society during his last visit to New York, in 1926, to the effect that if he knew how to produce hypercementosis, he would have done it for himself, his wife, his friends, and his patients. In view of these conflicting opinions regarding the nature of hypercementosis, and its clinical importance as an oral pathological condition that causes serious systemic

[1] Italic not in the original.

FIG. 11

FIG. 12

FIG. 13

FIG. 14

FIG. 15

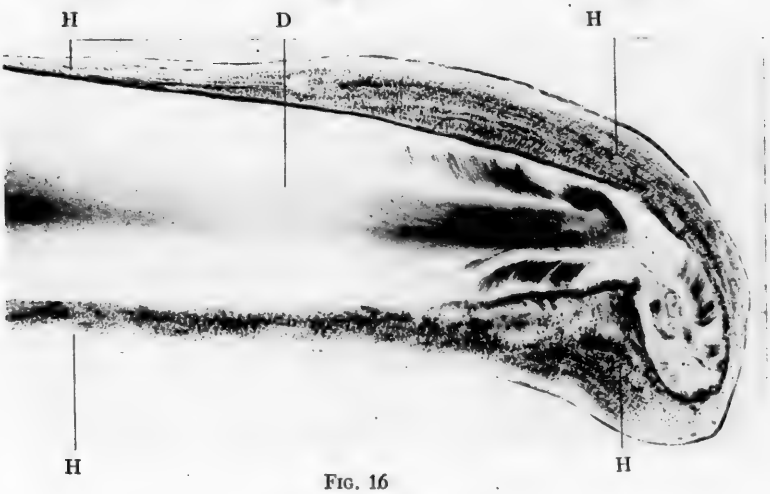

FIG. 16

FIGS. 11–16. CASE I

C—cementum. *D*—dentin. *E*—enamel. *H*—hypercementosis. *P*—pulp.

Fig. 11. Roentgenogram revealing presence of unerupted and malposed left maxillary canine.

Fig. 12. Roentgenogram of same area as that in *fig. 11* after removal of unerupted tooth.

Fig. 13. Photograph of malposed tooth removed from case shown in *fig. 11*.

Fig. 14. Roentgenogram of removed and malposed tooth shown in *figs. 11* and *13*.

Fig. 15. Roentgenogram of specimens made from tooth shown in *figs. 11, 13* and *14*.

Fig. 16. Low-power photomicrograph of ground section prepared from unerupted tooth shown in *figs. 11, 13, 14* and *15*. *Shows marked hypercementosis at apical third.*

FIG. 17

FIG. 18

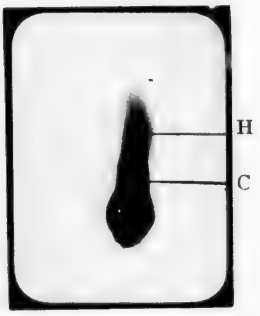

FIG. 19

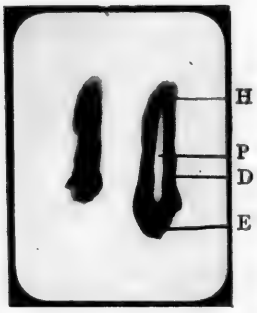

FIG. 20

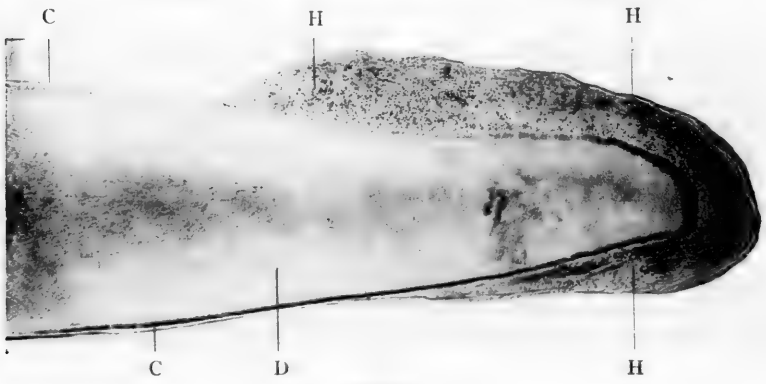

FIG. 21

FIGS. 17–21. CASE II

C—cementum. *D*—dentin. *E*—enamel. *H*—hypercementosis. *P*—pulp.

Fig. 17. Roentgenogram of left maxillary central, lateral and deciduous canine and premolars, revealing presence of unerupted and impacted canine.

Fig. 18. Photograph of unerupted and impacted canine shown in *fig. 17*.

Fig. 19. Roentgenogram of unerupted and impacted canine shown in *figs. 17* and *18*. See bulbous portion of root; due to cement hyperplasia covering mesial surface of apical half of root.

Fig. 20. Roentgenogram of sections of unerupted and impacted canine shown in *figs. 17*, *18* and *19*.

Fig. 21. Low-power photomicrograph of ground section of unerupted, malposed, canine shown in *figs. 17* and *18*. *Shows marked hypercementosis involving mesial aspect of apical half and apical third of entire root.*

FIG. 22

FIG. 23

FIG. 24

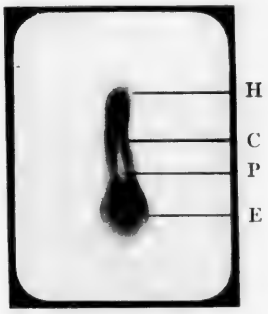

FIG. 25

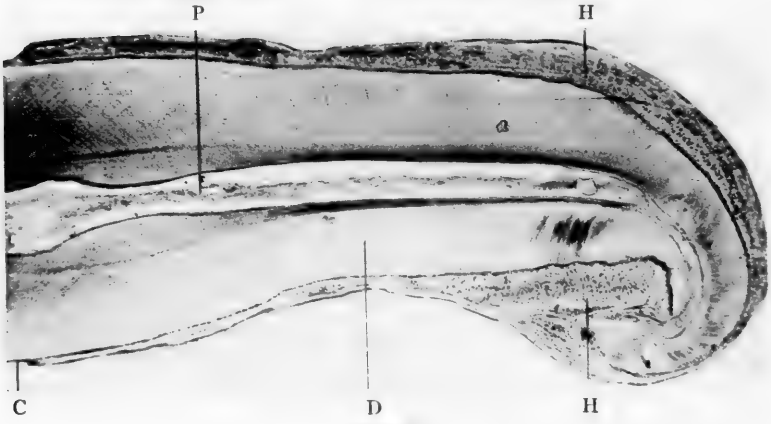

FIG. 26

FIGS. 22–26. CASE III

C—cementum. *D*—dentin. *E*—enamel. *H*—hypercementosis. *P*—pulp.

Fig. 22. Roentgenogram of region of left maxillary canine and bicuspids, revealing presence of unerupted and malposed maxillary left canine.

Fig. 23. Roentgenogram of same area as that in *fig. 22*. Taken after removal of impaction.

Fig. 24. Photograph of unerupted and malposed canine shown in *fig. 22*.

Fig. 25. Roentgenogram of unerupted and malposed maxillary canine removed from patient shown in *fig. 22*. See bulbous portion of apical third of root; due to presence of hypercementosis.

Fig. 26. Low-power photomicrograph of ground section of unerupted and malposed maxillary canine shown in *figs. 22, 24* and *25*. *Shows marked hypercementosis.*

disturbances, a further investigation is necessary. Accordingly, the present work was started with a study of material heretofore given comparatively little attention. I refer to hypercementosed, malposed, unerupted teeth. So far as I know, Kronfeld (8) has published the only paper dealing with this aspect of the subject. I shall have occasion to refer to it again. The occurrence of hypercementosis on unerupted and malposed teeth[2] seems to indicate quite definitely that this structure (hypercementosis) is not caused by infection, for some unerupted and malposed teeth that are free from pathological conditions present marked hypercementosis.

This report represents a brief account of a study of only five cases of unerupted and malposed teeth. It does not attempt to draw any *definite* conclusion and represents, as the title indicates, only a preliminary report of findings based upon a study of material taken from a source different from that investigated by previous writers. The reason for presenting a report of an *incomplete* investigation is to be found in Dr. Bödecker's plea "for more concerted action in the field of scientific research work." In an address delivered before the Pathodontia Section of the First District Dental Society of the State of New York (1923), Dr. Bödecker said: "Up to the present time, each man who had an aptness for this field did his work often in spare time only, as a hobby, and after years of labor would publish his results in a journal. These findings would often remain for years unrefuted or uncorroborated until some other lonely soul would happen to do some research work along similar lines. A controversy would ensue, usually through the medium of some journal, stretching over a great space of time and definite conclusions would be reached but slowly, the profession at large reaping but slight or no benefit whatever" (1). I could have continued this investigation and, as Bödecker says, after years of labor could have published my results, but in such a case the findings, as he rightfully indicates, would have remained for years *unrefuted* or *uncorroborated*. To pursue such a course in these days is obviously unwise, and for the following reason: teeth are being extracted incessantly. Many are removed because they present some very definite pathological condition, that is, evidence of periodontal disease or periapical infection. The sacrifice of such

[2] These represent about 10 per cent of all unerupted teeth removed by the author.

teeth is justifiable, but other teeth are being extracted on grounds that do not seem justifiable. Among these teeth are those exhibiting hypercementosis. Removal of such teeth, because some writers *believe* that they are pathological, is not warranted.

The authors of the old text-books as well as of those recently published, who consider hypercementosis pathological, are men of enviable reputation. It is true, also, that the writers who claim that serious disturbances are due to, and are relieved by extraction of, hypercementosed teeth are absolutely in earnest. But there is a deplorable situation in this connection that requires attention. Endelman and Wagner (5), in their treatise on general and dental pathology (1920), say regarding hypercementosis that it "*may be the etiologic factor of serious nervous manifestations.*"[3] Burchard and Inglis (4), in their book on dental pathology and therapeutics (1915), referring to hypercementosed teeth, said: "*Neuralgia, functional blindness, functional deafness, chorea, epileptiform fits, paralysis, cardiac neuralgia, insanity and other related conditions have been cured by the extraction of hypercementosed teeth.*"[3] This, the authors state, is based upon a statement by Brubaker (2) published in the American System of Dentistry (1887). Bunting, in his recent work on oral pathology (1929), wrote that "these cemental overgrowths may produce low-grade irritation in the surrounding tissues. They usually are not registered as a disturbance of the tooth affected, but more often give rise to reflected and diffuse pain and discomfort, the source of which is often difficult to locate. Thus neuralgic disturbances of various types may arise which are relieved by the extraction of the teeth so affected. *Cases have been reported in which serious nervous disturbances such as chorea, epilepsy, paralysis and insanity have been traced to dental hypercementosis and have been relieved by the extraction of such teeth. . . . . .*" (Italic not in the original.)

A comparison of these excerpts referring to hypercementosis seems to indicate that they were derived from one and the same source; namely, Brubaker's statement as published in the American System of Dentistry. The views of that author have obviously been taken at their face value and, without any attempt at corroboration, reprinted by the succeeding authors. There are numerous examples in our

[3] Italic not in the original.

FIG. 27

FIG. 28

FIG. 29

FIG. 30

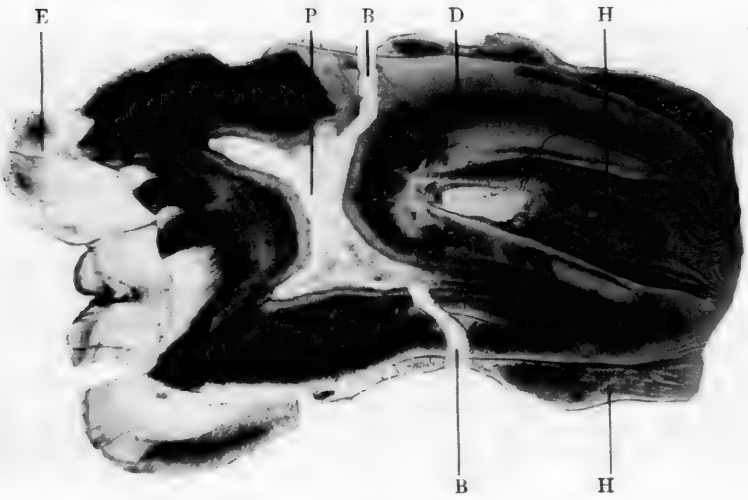

FIG. 31

FIGS. 27–31. CASE IV

B—Break in specimen. *D*—dentin. *E*—enamel. *H*—hypercementosis. *P*—pulp.

Fig. 27. Roentgenogram of region of lower left third molar, revealing presence of unerupted and malposed tooth in course of mandibular canal.

Fig. 28. Photograph of removed, malposed, unerupted left third molar shown in *fig. 27*.

Fig. 29. Roentgenogram of malposed and unerupted molar, shown in mesio-buccal aspect.

Fig. 30. Roentgenogram of tooth in *fig. 29*—mesio-distal view—showing ***marked hypercementosis.***

Fig. 31. Low-power photomicrograph of section of tooth in *fig. 30*. ***Shows marked hypercementosis.***

literature definitely showing the tendency of writers to take a statement by one investigator and, without corroboration, to repeat it again and again, usually dressed up to look like their own, until it finds its way into text-books and reference works and thus becomes accepted as an established fact. The effects of this unscientific procedure are obvious.

It seems evident, therefore, that an earnest effort should be made to determine definitely the status of hypercementosed teeth. By publishing this report it is hoped to stimulate the interest of other investigators and possibly to establish a contact with, using Dr. Bödecker's expression, "some other lonely soul" who "would happen to do some research work along similar lines." It may be possible that "more concerted action" may thereby be effected. The results obtained could be either refuted or corroborated without "stretching over a great period of time." It may also be possible, as Dr. Bödecker has said, that "definite conclusions would be reached" real soon and that "the profession at large may thus reap the benefit."

II. REPORT OF CASES AND FINDINGS

Thus far five cases have been studied. The ages of the patients treated ranged from 24 to 46 years. None of these patients presented any clinical symptoms, and the discovered unerupted, malposed, or impacted teeth were removed purely as a prophylactic measure. Brief histories of the cases studied and the histological findings are appended.

Case I: Peter S., 44 years of age. In routine roentgenographic examination to determine condition of teeth, presence of unerupted, malposed, left maxillary cuspid discovered. Tooth removed as prophylactic measure. Roentgenograms of unerupted, malposed, cuspid before and after removal, and those of sections made together with photographs and photomicrographs of specimens prepared from tooth, shown in *figs. 11* to *16*.

Case II: Barbara De P., 26 years of age; presented with slightly carious, deciduous maxillary canine. She desired to ascertain whether condition of tooth required removal. Roentgenographic examination (*fig. 17*) disclosed unerupted and malposed canine. Patient, having heard of disturbances that arise at times from unerupted and malposed teeth, submitted herself for removal of tooth. See *figs. 18* and *19*. Specimens prepared from tooth shown in *figs. 20* and *21*. *Figs. 18, 19,* and *21* show presence of

FIG. 32

FIG. 33

FIG. 34

FIGS. 32–34. CASE V

C—cementum. *D*—dentin. *E*—enamel. *H*—hypercementosis. *P*—pulp.

Fig. 32. Roentgenogram of left maxillary molars, revealing presence of unerupted and malposed maxillary third molar.

Fig. 33. Roentgenogram of sections of tooth shown in *fig. 32*.

Fig. 34. Low-power photomicrograph of ground section made from unerupted and malposed maxillary third molar in *figs. 32* and *33*. *Shows marked hypercementosis involving entire root.*

marked hypercementosis; no indication of it in *fig. 17.* Tooth presented no signs of any pathological condition.

Case III: Irving K., 33 years of age; consulted his dentist regarding stationary bridge which had become loose. To ascertain condition of teeth supporting bridge, patient was asked to have mouth roentgenographed. Unerupted and malposed impacted left maxillary canine (*fig. 22*) among existing pathological conditions of mouth. Dentist suggested removal of malposed, impacted tooth before new bridge constructed. Following this advice, patient submitted himself for removal of impaction, which proved to be perfectly healthy tooth presenting *marked hypercementosis.* See *figs. 22–26.*

Case IV:[4] Woman, 52 years of age; extreme nervousness chief complaint. Physicians she consulted advised thorough mouth examination with hope of finding cause or contributory factor in patient's sufferings. Roentgenographic examination of teeth and jaws disclosed, among various pathological conditions, unerupted and malposed mandibular third molar (*fig. 27*). Removed; had *marked hypercementosis.* (See *figs. 27–31.*)

Case V: Mrs. Anna J., 34 years of age; told she had maxillary sinusitis, came to writer with request for thorough mouth examination. To test diagnosis and determine presence of any oral pathological conditions, ensuing roentgenographic examination revealed unerupted and malposed left maxillary third molar. Removed, and sectioned (*figs. 32–34*).

III. SUMMARY

A condition characterized by an excessive growth of cementum, which is easily recognized on certain extracted teeth and roentgenograms taken of them, is called hypercementosis. This condition has been found associated with both healthy and diseased teeth. The cause of hypercementosis has not been determined. Opinions regarding this condition differ: some maintain that it is beneficial; others, that it is pathological and should be eradicated. Thus, the status of hypercementosed teeth is uncertain. As a consequence, many hypercementosed teeth are being sacrificed. This situation warrants further study of the problem.

The present writer suggests that all who may be engaged in the study of hypercementosis should coöperate, and report all their findings while their work is being conducted.

[4] Case of M. Lentz, D.D.S., Passaic, N. J.

The report herein presented is a study of five cases of unerupted, impacted, and malposed teeth presenting no evidence of any pathological condition. The microscopical and clinical examinations of these teeth reveal the presence of marked hypercementosis involving various portions of the root.

IV. GENERAL CONCLUSION

Hypercementosis having been found on unerupted, impacted, and malposed teeth, which presented no pathological signs, this condition cannot be due, in all cases, to infection as some writers believe, and therefore cannot always be considered pathological.

I am greatly indebted to Prof. Charles F. Bödecker for his friendly encouragement and guidance in this research. To Dr. Edmund Applebaum, who aided me in the preparation of the photomicrographs, I wish to acknowledge my indebtedness.

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355 East 149th Street,
New York City

Quotations Relating to "Dentistry or Stomatology"

THIS JOURNAL uses the term "dentistry" to signify the science and art of oral health service in all its local and systemic relationships. The meanings of words, which are not necessarily restricted to *etymological* significance, acquire expanded or new import in accordance with convenience and usage. Thus the term "medicine," which originally meant to remedy or cure, now also means *to make remedial or curative treatment unnecessary.* "Stomatology" is a synonym for "dentistry." We prefer the older term because its use respects both the history of dentistry and the efforts of those who have brought dentistry to its present status.—*Jour. of Dental Research*, 1929, ix, p. iii.

If dentistry were merely dental mechanics, a two-year course in dental technology, based on graduation from a high school and conducted along lines similar to those for the education of optometrists, would probably afford sufficient training for dental practice, and oral health-service could be reserved for a new specialty to include oral surgery, which might be named stomatology and made analogous to ophthalmology and other specialties of medicine. But with dentistry developed into the *full service equivalence* of an oral specialty of medicine, the creation of a medical specialty of stomatology would be redundant—and there would be no occasion to substitute "stomatology" for the name established by custom.—*Carnegie Foundation's Bulletin on Dental Education*, 1926, p. 135.

The assumption that dentistry would be something more or better than it is, if all or a part of it were called "stomatology," exemplifies a readiness to attach more importance to labels than to contents, or more significance to make-believe than to reality. The term "medicine" originally meant to remedy or cure. ["*Medicina* (ars)"—healing art; "*medicina* (res)"—a remedy; "*medicus*"—of healing; also a physician.] Today "medicine," by including measures to *prevent* sickness and by throwing *etymology* overboard, means not only to remedy or cure, but also *quite the contrary*—to make remedial or curative treatment unnecessary. As medical knowledge has advanced and medical procedures have been changed in consonance, including the development of prevention and of surgery, new meanings have been imparted to the term "medicine." But instead of replacing "medicine" by a succession of words intended to indicate *etymologically* the broader or deeper modern meanings, the import of the original term has been conveniently enlarged as the scope of medical practice has expanded. Thus, the term "medicine" has come to mean more and more as *physicians*, without restricting themselves to its *etymology*, have included *more and more in its significance.* Along the same line of reasoning one sees clearly that, although *etymologically* the term "dentistry" refers only to the teeth, nevertheless logically it also comprehends the contiguous parts and their systemic relationships. Certainly by *usage* in conformity with this logic, and also with the criteria of common sense, the term "dentistry" has *acquired* the broader meaning suggested by the more detailed and lengthy term "oral health-service." By logic, usage, and convenience, the term "dentistry" has thus become the *practical* equivalent of "stomatology," which *etymologically* means the sum of scientific knowledge concerning the mouth, but *etymologically does not include the rest of the body* (although "stomatology" is now *used* by some to include systemic relationships with the mouth). This being true, we are unable to agree that "dentistry" and "dentists," which are terms that have long represented the earnest and aspiring endeavors of generations of faithful practitioners of oral health-service, should be formally replaced for *etymological* reasons by "stomatology" and "stomatologists."—*American College of Dentists*, March, 1929.

The New York Academy of Dentistry . . . without any diminution of its abiding purpose to promote intimate association and cooperation between dentists and the practitioners of all other kinds of health-service, believes that, in the interest of the patient, dentistry should be *continued* as a separately organized profession, based on systems of ethics, education, jurisprudence, and journalism that will fully meet all public needs at each stage of its evolution.—*N. Y. Academy of Dentistry, April, 1929.*

The Dental Society of the State of New York . . . expresses the earnest conviction . . . that the most effectual development of oral health-service will be insured by the *continuance* of dentistry as a separately organized profession . . . [and] also records its unabated desire and intention to promote personal and professional accord, cooperation, and mutual helpfulness between dentists and the practitioners of medicine and all other kinds of health-service.—*Dental Society of the State of New York, May, 1929.*

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